Nathaniel M. Cohen
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# DISEASES OF CHILDREN

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## A PRACTICAL TREATISE ON DIAGNOSIS AND TREATMENT FOR THE USE OF STUDENTS AND PRACTITIONERS OF MEDICINE

BY

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TO

MY WIFE,

WHO HELPED ME WRITE THIS BOOK

# PREFACE

In this volume the author has attempted to present to practitioners and students of medicine a practical clinical treatise on diseases of infants and children. He has but briefly outlined the pathological findings and has avoided unnecessary etiological discussions in order that he might, in a compact volume, find more space in which to clearly outline the differential diagnosis and give in full the treatment of these diseases.

The diseases of infancy and childhood differ very widely in their clinical manifestations and in the methods of their successful treatment from corresponding diseases in the adult; the reasons for this are physiological rather than pathological. The undeveloped organism of the child, because of its physiological peculiarities, reacts to the same pathological factors very differently from the completed and stable organism of the adult. For this reason the author has dwelt in detail on the physiological factors of disease in infancy and childhood, and has attempted to make practical application of these facts both in the prophylactic and curative treatment of these diseases.

The author acknowledges his indebtedness to Gen. Wm. M. Wherry, Dr. M. A. Brown, Dr. Alfred Friedlander, Dr. David I. Wolfstein, Dr. Max Dreyfoos, Dr. Frank H. Lamb, Dr. M. L. Heidingsfeld, and Dr. Samuel Iglauer for revision of manuscript, and to Dr. W. J. Graf for photographic work.

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# CONTENTS

## SECTION I

## THE CHILD


CHAPTER PAGE

I. General Hygiene of Infancy and Childhood . . 1
Chief causes of death in infancy and childhood; general hygiene and care of infants and young children.

II. Growth and Development . . . . . 13
Weight during infancy and early childhood; height of child at different ages; head measurements; development of the spine and bony framework; muscular development; the special senses; the nervous system; the heat-regulating mechanism.

III. Examination of the Sick Child . . . . . 28
Present illness; previous medical history; family history; physical examination; inspection; palpation; reflexes; percussion; auscultation; macroscopic and microscopic examination of intestinal discharges; tuberculin skin reactions; blood examinations; lumbar puncture; radiography.

IV. Therapeutics of Infancy and Childhood . . . 38
Drug administration by the mouth; inunctions; fresh air; hydrotherapy; hypodermoclysis; nasal douche; stomach-washing; rectal enemata; rest-cure; massage; active and passive exercises; psychotherapy; vaccine therapy.


## SECTION II

## THE NEW-BORN


V. The Care of Premature Infants . . . . . 62
Physiological peculiarities of the new-born; incubator; padded basket; feeding.

VI. Diseases of the New-Born . . . . . 69
Asphyxia neonatorum; congenital atelectasis.

VII. Diseases of the New-Born (Continued) . . . 75
Septic infection; dermatitis exfoliativa; erysipelas;

CHAPTER PAGE

hemorrhages in the new-born; diseases of the umbilicus; mastitis; Holt's inanition fever.

VIII. Diseases of the New-Born (Continued) . . . 89
Tetanus neonatorum; icterus neonatorum; occlusion of the bile ducts in the new-born; other forms of icterus occurring in the new-born; ophthalmia neonatorum.

IX. Birth Injuries . . . . . 97
Cephalhematoma; hematoma; birth palsies.

SECTION III

INFANT FEEDING

X. Milk in Its Relation to Infantile Nutrition . . 100
Composition; digestibility, and relative importance of the various ingredients of milk.

XI. Human Breast Milk in Its Relations to Infant Feeding . . . . . 107
Composition of colostrum and human milk; how to determine wholesomeness of breast milk; how to modify its quantity and quality.

XII. Breast-Feeding . . . . . 112
Principles underlying normal breast-feeding; mixed feeding; weaning; the wet-nurse.

XIII. Food Materials Used in the Artificial Feeding of Infants . . . . . 118
Clean raw cow's milk; sterilized milk; pasteurized milk; peptonized milk; buttermilk; Finkelstein's albumin milk; skim milk; whey; carbohydrates; proprietary foods; albumin water; meat preparations.

XIV. Artificial Feeding . . . . . 129
Value of percentage feeding; calorimetric standard; principles underlying artificial feeding; home modification of milk; value of carbohydrate and alkaline diluents; feeding of difficult cases; laboratory method of modifying milk; diet of children as they grow older.

SECTION IV

DISEASES OF THE DIGESTIVE SYSTEM

XV. Dentition . . . . . 144
The eruption of the temporary teeth; dentition as a pathological factor; permanent teeth.

CHAPTER PAGE

XVI. Stomatitis . . . . . 146
Stomatitis catarrhalis; stomatitis aphthosa; stomatitis mycosa; stomatitis ulcerosa; stomatitis gangrenosa.

XVII. Other Diseases of the Mouth and Diseases of the Esophagus . . . . . 154
Bednar's aphthæ; perlèche; elongated uvula; geographical tongue; tongue-tie; hare-lip; esophagitis; periesophageal abscess; branchial cysts.

XVIII. Diseases of the Stomach . . . . . 158
Acute gastric indigestion; acute gastritis; dilatation of the stomach; ulcer of the stomach; acute gastroduodenitis; congenital hypertrophy of the pylorus; chronic gastritis.

XIX. Etiology and Preventive Treatment of the Intestinal Disorders of Infancy . . . . . 173

XX. Acute Intestinal Indigestion . . . . . 183
Food injuries; fat indigestion; sugar indigestion; protein indigestion.

XXI. Enteric Infection . . . . . 189
Acute enterocolitis; cholera infantum.

XXII. Chronic Intestinal Indigestion . . . . . 197
Chronic enterocolitis; infantile atrophy; marasmus; chronic intestinal indigestion in older children.

XXIII. Chronic Constipation in Infancy and Congenital Dilatation of the Colon . . . . . 204

XXIV. Intestinal Parasites . . . . . 210
Tape-worms; round-worms; thread-worms.

XXV. Intestinal Intussusception . . . . . 219

XXVI. Appendicitis . . . . . 222

XXVII. Peritonitis and Ascites . . . . . 228

XXVIII. The Rectum and the Anus . . . . . 233
Malformations; polypi; prolapse; fissure of anus.

## SECTION V

## NUTRITIONAL DISORDERS

XXIX. Rickets . . . . . 237

XXX. Infantile Scurvy . . . . . 246

XXXI. Diabetes Mellitus . . . . . 250

XXXII. Recurrent Vomiting, Recurrent Coryza and Migraine 251
Severe acid intoxication with gastroenteritis; recurrent coryza; migraine.

## SECTION VI

## INFECTIOUS DISEASES


| CHAPTER | | PAGE |
|---|---|---|
| XXXIII. | Fever | 260 |
| XXXIV. | Typhoid Fever | 267 |
| XXXV. | Malaria | 279 |
| XXXVI. | Whooping Cough | 287 |
| XXXVII. | Diphtheria | 295 |
| XXXVIII. | Influenza | 308 |
| XXXIX. | Scarlet Fever | 315 |
| XL. | Measles<br>Rubella, erythema infectiosum. | 332 |
| XLI. | Variola<br>Vaccinia; varicella. | 345 |
| XLII. | Mumps | 357 |
| XLIII. | Syphilis | 360 |
| XLIV. | Tuberculosis | 373 |
| XLV. | Acute Articular Rheumatism and Other Forms of Arthritis | 402 |


## SECTION VII

## DISEASES OF THE RESPIRATORY SYSTEM


| | | |
|---|---|---|
| XLVI. | Diseases of the Nasal Mucous Membrane<br>Rhinitis; epistaxis; foreign bodies in the nose. | 411 |
| XLVII. | Diseases of Tonsils<br>Acute follicular tonsillitis; Vincent's angina; chronic tonsillar hypertrophy; quinsy. | 417 |
| XLVIII. | Diseases of Pharynx<br>Adenoids; acute retropharyngeal abscess. | 424 |
| XLIX. | Diseases of Larynx<br>Acute laryngitis; edema of the larynx; growths and foreign bodies in the larynx; congenital laryngeal stridor. | 429 |
| L. | Bronchitis<br>Acute catarrhal bronchitis; chronic bronchitis; membranous bronchitis. | 438 |
| LI. | Lobar Pneumonia | 442 |
| LII. | Bronchopneumonia | 454 |
| LIII. | Pleurisy | 470 |

## SECTION VIII

## THE HEART


| CHAPTER | | PAGE |
|---|---|---|
| LIV. | Congenital Heart Disease | 484 |
| LV. | Acute Endocarditis | 489 |
| LVI. | Myocarditis and Acute Cardiac Dilatation | 494 |
| LVII. | Chronic Valvular Disease | 498 |
| LVIII. | Functional Cardiac Disorders | 506 |
| LIX. | Pericarditis | 509 |


## SECTION IX

## DISEASES OF THE BLOOD AND DUCTLESS GLANDS


| | | |
|---|---|---|
| LX. | The Blood | 515 |
| LXI. | Simple Secondary Anemia | 522 |
| LXII. | Pseudo-Leukemia of Infants | 524 |
| LXIII. | Chlorosis | 526 |
| LXIV. | Pernicious Anemia | 528 |
| LXV. | Leukemia | 530 |
| LXVI. | Purpura | 531 |
| LXVII. | Hemophilia | 537 |
| LXVIII. | Hodgkin's Disease | 540 |
| LXIX. | Simple Adenitis | 542 |
| LXX. | Status Lymphaticus | 545 |
| LXXI. | Diseases of the Spleen | 551 |
| LXXII. | Diseases of the Thyroid Gland<br>Sporadic cretinism. | 552 |


## SECTION X

## DISEASES OF THE UROGENITAL SYSTEM


| | | |
|---|---|---|
| LXXIII. | The Urine<br>Lithuria; indicanuria; hematuria; hemoglobinuria; acetonuria; physiological albuminuria; orthostatic albuminuria. | 559 |
| LXXIV. | Acute Nephritis | 571 |
| LXXV. | Chronic Nephritis and Other Diseases of the Kidneys<br>Chronic diffuse nephritis; cystopyelitis; tumors of the kidney; hydronephrosis; perinephritis; dislocation of the kidney. | 582 |

CHAPTER PAGE

LXXVI. Diseases of the Genital Organs . . . . . 591
Gonorrheal vulvovaginitis; simple vulvovaginitis; urethritis; adherent prepuce; phimosis; periphimosis; undescended testicle; hypospadias; enuresis.

LXXVII. Pseudomasturbation in Infants . . . . . 603

## SECTION XI

## DISEASES OF THE NERVOUS SYSTEM

LXXVIII. Diseases of the Brain . . . . . 610
Infantile cerebral palsies; brain tumors; abscess of the brain; chronic internal hydrocephalus; meningocele; encephalocele; hydrencephalocele; idiocy.

LXXIX. Meningitis . . . . . 626
Tuberculous meningitis; meningococcus meningitis; purulent meningitis.

LXXX. Diseases of the Spinal Cord . . . . . 641
Anterior poliomyelitis; myelitis; hereditary ataxia; spina bifida.

LXXXI. Diseases of Peripheral Nerves . . . . . 661
Multiple neuritis; facial paralysis; progressive muscular dystrophy.

LXXXII. General Nervous Diseases . . . . . 669
Disorders of sleep; eclampsia in infants and children; laryngismus stridulus; tetany in infancy and childhood; nystagmus; epilepsy; chorea; habit-spasm; pica; hysteria; headaches; asthma.

## SECTION XII

## DISEASES OF THE EAR

LXXXIII. Otitis Media and Mastoiditis . . . . . 714

## SECTION XIII

## DISEASES OF THE SKIN

LXXXIV. Eczema and Other Skin Diseases . . . . . 719
Eczema; urticaria; furunculosis; erythema multiforme; congenital ichthyosis; impetigo contagiosa; pemphigus neonatorum; tinea tonsurans; scabies; pediculosis capillitii.

# LIST OF ILLUSTRATIONS

## PLATES


| PLATE | | FACING PAGE |
|---|---|---|
| I. | The Moro tuberculin skin reaction | 36 |
| II. | Blood picture in dermatitis exfoliativa | 79 |
| III. | The enanthem of measles (Koplik's spots) | 335 |
| IV. | The von Pirquet tuberculin skin reaction | 385 |
| V. | Scheme of the circulation of the blood in the fetus | 483 |
| VI. | Blood pictures in von Jaksch's disease, and acute lymphatic leukemia | 525 |


## ILLUSTRATIONS IN THE TEXT


| FIGURE | | PAGE |
|---|---|---|
| 1. | Effect of electrical stimulation on the spinal ganglion of cat | 12 |
| 2. | The rate of growth in weight of dull, mediocre, and precocious boys and girls | 16 |
| 3. | Bad position while writing | 20, 21 |
| 4. | Palpating spinal curvature | 32 |
| 5. | Percussion and auscultation position | 33 |
| 6. | Position for lumbar puncture | 37 |
| 7. | Fresh-air ward | 44 |
| 8. | Hypodermoclysis | 47 |
| 9. | Position for nasal douching | 48 |
| 10. | Stomach-washing | 50 |
| 11. | Temperature curve showing influence of artificial heat on premature infant | 63 |
| 12. | Padded basket for treatment of premature infants | 67 |
| 13. | Breck's feeding-tube | 68 |
| 14. | Schultze's method of artificial respiration | 72 |
| 15. | Blood chart in dermatitis exfoliativa | 79 |
| 16. | Umbilical hernia | 86 |
| 17. | Colostrum | 108 |
| 18. | Woman's milk | 108 |
| 19. | Weight chart breast-fed infant | 114 |
| 20. | Freeman's pasteurizer | 121 |
| 21. | Weight chart artificially fed infant | 136 |
| 22. | Thrush fungus | 149 |
| 23. | Stomatitis gangrenosa, before perforation | 152 |

| FIGURE | | PAGE |
|---|---|---|
| 24. | Stomatitis gangrenosa, after perforation | 152 |
| 25. | Radiograph, pyloric stenosis | 166 |
| 26. | Congenital stenosis of the pylorus | 167 |
| 27. | Congenital stenosis of the pylorus, longitudinal section through tumor mass | 169 |
| 28. | Temperature chart, gastroenteric infection, mild | 189 |
| 29. | Temperature chart, gastroenteric infection, severe | 190 |
| 30. | Casein curds | 198 |
| 31. | Infantile atrophy | 199 |
| 32. | Tænia saginata | 211 |
| 33. | Tænia solium | 212 |
| 34. | Ascaris lumbricoides | 216 |
| 35. | Egg of ascaris lumbricoides | 216 |
| 36. | Oxyuris vermicularis | 218 |
| 37. | Acute peritonitis position | 230 |
| 38. | Malformations of the rectum | 233 |
| 39. | Radiograph showing bony deformities in rickets | 238 |
| 40. | A case of rickets | 239 |
| 41. | Knock-knees and bow-legs | 241 |
| 42. | Temperature chart, typhoid fever, child 2½ years of age | 270 |
| 43. | Temperature chart, typhoid fever, child 6 years of age | 271 |
| 44. | Temperature chart, typhoid fever, child 10 years of age | 272 |
| 45. | Temperature chart, typhoid fever with relapses | 274 |
| 46. | Temperature chart, malarial fever | 282 |
| 47. | Temperature chart, pertussis and measles complicated by bronchopneumonia | 290 |
| 48. | Temperature chart, pharyngeal diphtheria treated with antitoxin | 297 |
| 49. | Temperature chart, laryngeal diphtheria treated by intubation | 298 |
| 50. | Chart showing decreased death rate from diphtheria under antitoxin | 303 |
| 51. | Intubation position | 306 |
| 52. | Temperature chart, scarlet fever, mild, child 12 years of age | 319 |
| 53. | Temperature chart, scarlet fever, severe, child 6 years of age | 320 |
| 54. | Chart showing mortality by age in 5,000 cases of scarlet fever | 326 |
| 55. | Temperature chart, measles, uncomplicated | 336 |
| 56. | Temperature chart, measles, complicated by bronchopneumonia; died | 337 |
| 57. | Temperature chart, measles, complicated by bronchopneumonia; recovered | 338 |
| 58. | Varicella eruption on the fourth day | 355 |
| 59. | Syphilitic dactylitis | 365 |
| 60. | "Hutchinson's teeth" | 366 |
| 61. | Bronchial and other lymph nodes mainly affected in tuberculosis | 379 |
| 62. | Radiograph, enlarged bronchial glands at right hilum | 380 |
| 63. | Pulmonary tuberculosis with left-sided pneumothorax | 389 |
| 64. | Ileopelvic lymphatic glands | 390 |

| FIGURE | | PAGE |
|---|---|---|
| 65. | Anterior view of the cecum and appendix | 391 |
| 66. | Tuberculosis of the spine | 392 |
| 67. | Position in examination for adenoid growths | 427 |
| 68. | Retropharyngeal lymph glands | 428 |
| 69. | Radiograph of foreign body in right bronchus | 436 |
| 70. | Temperature chart, lobar pneumonia, child 2 years of age | 445 |
| 71. | Temperature chart, lobar pneumonia, child 4 years of age | 446 |
| 72. | Temperature chart, lobar pneumonia, child 10 years of age | 447 |
| 73. | Temperature chart, bronchopneumonia, mild | 459 |
| 74. | Temperature chart, bronchopneumonia, severe | 460 |
| 75. | Temperature chart, empyema following pneumonia | 473 |
| 76. | Temperature chart, empyema | 474 |
| 77. | Radiograph, pleural effusion in chest | 476 |
| 78. | James' apparatus for expanding the lung | 481 |
| 79. | Clubbing of the fingers in congenital heart disease | 486 |
| 80. | Radiograph of enlarged heart from mitral regurgitation | 500 |
| 81. | Radiograph of pericarditis with effusion | 511 |
| 82. | Diagram showing blood changes in a case of purpura hemorrhagica | 534 |
| 83. | Position in palpating the spleen | 551 |
| 84. | Typical cretin, age 4 years | 554 |
| 85. | Same cretin after eight years of treatment | 555 |
| 86. | Sarcoma of the kidney | 588 |
| 87. | Genitourinary organs, embryo about five weeks | 604 |
| 88. | Genitourinary organs, embryo about nine weeks | 605 |
| 89. | Genitourinary organs | 606 |
| 90. | Hemiplegia from cerebral hemorrhage | 612 |
| 91. | Spastic diplegia | 614 |
| 92. | Idiopathic hydrocephalus | 619 |
| 93. | Meningocele, encephalocele, hydrencephalocele | 621 |
| 94. | Temperature chart, tuberculous meningitis | 629 |
| 95. | Opisthotonos | 633 |
| 96. | Temperature chart, meningococcus meningitis | 634 |
| 97. | Temperature chart, pneumococcus meningitis | 640 |
| 98. | Temperature chart, acute anterior poliomyelitis | 646 |
| 99. | Deformities resulting from acute anterior poliomyelitis | 647 |
| 100. | Quadrupedal gait from acute anterior poliomyelitis | 648 |
| 101. | Hydrencephalocele and spina bifida | 659 |
| 102. | Progressive muscular dystrophy | 667 |
| 103. | Progressive muscular dystrophy | 668 |
| 104. | Progressive muscular dystrophy | 669 |
| 105. | Impetigo contagiosa | 731 |
| 106. | Ringworm of the scalp | 733 |
| 107. | Pustular scabies of the hands | 736 |

# PEDIATRICS

## SECTION I

## *THE CHILD*

### CHAPTER I

#### GENERAL HYGIENE OF INFANCY AND CHILDHOOD

### CHIEF CAUSES OF DEATH IN INFANCY AND CHILDHOOD

The importance of the proper care of infants and young children is emphasized by the terrible mortality which occurs during the early months and years of life, and by the fact that this mortality is to a large extent due to remediable causes pertaining to general hygiene. Only a few years ago it was estimated that about 25 per cent. of all infants living in the large cities of the world died during the first year of life, and of these deaths from 20 to 25 per cent. occurred during the first month of life. After the first month there is a sudden fall in the death rate, and thereafter it slowly decreases throughout the year. The mortality after the first year of life continues high, but gradually diminishes up to the fifth year of life. At this period there is a second rapid fall in the death rate, but the mortality is still high as compared with that of the young adult and is gradually reduced up to the fifteenth year.

Holt says: "The fundamental causes of infant mortality are mainly the result of three conditions, poverty, ignorance and neglect." It is a matter of common observation that the great death rate among infants in our large cities occurs largely among the very poor. Among the well-to-do classes the infant mortality is comparatively slight.

The chief causes of death during the first year of life are prematurity, congenital debility from hereditary causes (syphilis, etc.), malformations, birth injuries, septic infection, whooping cough, gastrointestinal and acute respiratory diseases. It is evident that a certain proportion of these deaths, especially those due to prematurity, congenital debility, malformations and birth injuries, cannot be prevented, and yet the loss of life from these causes could be materially diminished by the proper medical care of the mother before, and of the infant directly after delivery. The mortality from septic infection and gastrointestinal diseases has been very materially

diminished in recent years by the improved hygienic methods for the care of the new-born.

Between the end of the first and the sixteenth month of life the high infantile mortality is largely kept up by gastrointestinal diseases, which could to a large degree be prevented if it were possible to give these unfortunate infants suitable food and place them under better hygienic surroundings.

After the end of the first year of life, gastrointestinal disorders still play an important part in producing the death roll; but from the end of the first to the fifth year of life influenza, bronchitis, and the pneumonias are the most important factors in keeping up the high death rate. These infectious respiratory diseases are largely air-borne and are promoted by overcrowding and unhygienic surroundings. They could therefore in great part be prevented if infants and young children could be separated from contagion and given pure flowing air to breathe.

After the fifth year the ordinary acute infections, such as scarlet fever, diphtheria, measles, and whooping cough, keep the mortality of later childhood higher than that of adult life. These diseases are also very much more prevalent in the unhygienic, crowded tenements of the poor than among the larger, well-ventilated homes of the middle and upper classes. The mortality at this time of life could therefore be very materially diminished by improving the facilities for the care and isolation of children suffering from these infectious diseases.

The fact that the great majority of deaths which occur among infants and young children could be prevented by suitable food, proper care, wholesome surroundings and protection from contagions has stimulated city health boards and co-operating philanthropical societies to attempt to apply these life-saving measures to the children of the poor in our large cities, and as a result of these efforts infantile mortality has been reduced from 26 to 15 per cent., and the mortality among children under five years of age has been reduced from 16 to 6 per cent. This remarkable showing of the influence which modern hygienic methods have had upon the saving of life should stimulate and encourage to still greater accomplishments all those interested in this great work.

## GENERAL HYGIENE AND CARE OF INFANTS AND CHILDREN

**Care of the New-Born.**—From what has been said it is evident that one of the most important duties of the physician is to keep children well, and this brings us to the consideration of the general hygiene of infancy and childhood.

Soon after birth the umbilical cord, when the pulsation has ceased, should be firmly tied with a piece of clean, narrow tape and then cut with clean scissors. Following this operation the infant's mouth should be washed out and its breathing and heart action carefully observed. If

these be normal and the infant has cried lustily, indicating that normal pulmonary inflation has begun, it may be wrapped for a few minutes in warm flannels, until the nurse who is attending the mother has the time to bathe it. The body of the infant should be gently rubbed with vaselin or olive oil to remove the vernix caseosa which covers its body. It should then be placed in warm water (temperature 100° F.) and gently washed with some non-irritating soap. The stump of the cord should be carefully dried and the surrounding parts dusted with talcum or some other powder; it should then be folded in a pad of sterile gauze. This may be done by making an opening in the pad through which the cord is inserted. Thereafter it is important to keep the cord, and the dressing which covers it, dry until mummification and separation take place; this usually occurs about the end of the first week. Following the separation of the cord the umbilicus for a few days presents a slightly red surface, over which the epithelium is rapidly forming. By the end of the second week the umbilical wound should be entirely covered with epithelium and should therefore no longer offer a favorable portal for septic infections. Until this occurs the infant is to be given one or more sponge baths every day, care being taken throughout the whole time to protect the umbilical wound from the wash water or other possible sources of infection. When the umbilical wound has healed the infant is to have a tub bath daily, beginning with a temperature of 100° F., gradually diminishing the temperature of the water as the child grows older, but during the first year of life it is not advisable that the temperature of this bath should fall much below 90° F. During the early days of the life of the infant it is important that the region of the umbilicus be examined without removing the dressing which holds the mummifying cord. A certain amount of redness in this region is normal, but if the parts become swollen, or if the odor from the cord becomes putrid, and especially if the temperature of the infant rises two or three degrees above normal without apparent cause, the dressing which covers the stump of the cord is to be carefully removed and evidences of sepsis looked for. In the event that the umbilical wound becomes infected, it is to be treated by the method described in the chapter on Sepsis in the New-Born.

In private practice it is, as a rule, only necessary to carefully wash out the eyes of the newly-born infant with distilled water, or a 3 to 5 per cent. boracic acid solution. But in the event that the mother has a vaginal discharge or the child is born in a public institution, it is advisable to instil into its eyes a 2 per cent. solution of nitrate of silver and thereafter carefully wash them out with sterile water. During the first days of life it is important, especially if the infant be irritable, to have its rectal temperature taken twice a day with a clean thermometer anointed with clean vaselin. A sharp elevation of temperature during the first days of life suggests either sepsis or Holt's (inanition) fever. It is most important during the first days of life to note the discharges from the gastrointestinal canal. The diapers containing these discharges should be saved for the inspection of the physician, as no one, not even a trained nurse, can ac-

curately convey to the physician their character. The early dark meconium discharges should commence to give way on the third day to milk stools, and within ten days or two weeks the fecal discharge should be gradually transformed into a soft, yellow, homogeneous mass. The appearance of dark, tarry stools after the fifth day is an indication of intestinal hemorrhage (melena), and the presence of mucus, curds and other abnormalities may be important early indications of an intestinal condition which needs attention.

**Rest and Sleep.**—The new-born should sleep nearly all the time, being awake but two or three hours in the twenty-four. A young infant, therefore, that spends much of its time awake, fretting and crying, is suffering from some condition which should be corrected; hunger, overfeeding and indigestion are common causes of fretfulness. As the infant grows older it is awake for longer periods of time, but even at one year of age it should sleep sixteen hours out of the twenty-four. It is most important during its waking hours that the infant should not be coddled and played with. It is a very difficult matter to enforce this rule. Most infants in the middle and upper walks of life are so surrounded by doting relatives that it is difficult to protect them from the incessant fondling and entertaining which these devoted and well-meaning people force upon them. As Northrup has so graphically pointed out, this is one of the most common causes of sleeplessness, irritability and nervousness in infants. For the good health and normal development of the infant it should be let alone during its waking hours. Properly trained babies are perfectly happy and will coo, and play with their toes or some other object which they happen to find, never knowing what it means to be taken up, dandled, coddled and entertained.

**Fresh Air.**—When, after a few weeks, the infant's nutritional problems have been solved and its heat regulating apparatus has been properly adjusted to surrounding conditions, it should be gradually accustomed to a temperature cooler than the ordinary house temperature. The windows of the room, little by little, should be opened, and the fresh air treatment which is to continue throughout childhood, and I might say throughout life, should be begun. Depending upon the season of the year, it is to be taken out of doors for a short or a long time each day, and windows are to be opened so that it shall have fresh and moderately cool air. As the infant becomes a child it should then live in pure, fresh air for the whole twenty-four hours. Open-air sleeping apartments and wide-open bedroom windows make this possible until school life begins, and then it is, as a rule, necessary that the child should be confined for a few hours during the day to the schoolroom, where the air is much less pure than out of doors and in homes and sleeping apartments. The transition from the coddling and warmth which are necessary during the first days of life to life in the open air throughout the twenty-four hours must be gradual. It is only when the child has reached the age of three years that it can be readily cared for in out-door sleeping apartments during the winter weather in our middle and northern states.

**The Nursery.**—Where it is possible, a large, bright, well-ventilated room should be selected for the nursery, as this is to be the indoor home of the child during the early years of its life. This room is to be devoted to the infant and its necessary attendant, and is not to be a reception room into which all interested relatives and visitors are ushered that they may observe a wonderfully precocious and beautiful baby. The young infant's undeveloped and excitable nervous system should be allowed to develop along normal lines and not be kept in a constant state of excitement and stimulation during waking hours. The nursery should be free from heavy rugs and hangings, as simply furnished as possible, and the air in it fresh, pure and free from contagion. As the natural instinct of the infant is to put everything into its mouth, its toys and other things with which it plays should be of such a character that they may be easily kept clean and the infant's surroundings should be such that these playthings will not be contaminated when they are dropped by its side. Rubber pacifiers should not be tolerated; their use results in an unhygienic habit which it is difficult to break. During the early months of life the infant should spend nearly all of its time on a flat mattress. It should not be encouraged to sit up or to stand upon its feet until its muscular and bony development are such that these procedures will not result in deformities such as curvature of the spine and bow-legs.

**Clothing.**—The young infant must be rather warmly clad, because its heat-regulating apparatus is not sufficiently developed to maintain a normal temperature under varying degrees of heat and cold. The laity, however, are thoroughly impressed with the fact that the newly-born infant requires more than the ordinary amount of clothing to keep it warm, and the tendency therefore is not only to clothe the infant too warmly, but to bundle it in such a manner as to interfere with the free expansion of its lungs and with the exercising of its arms and legs. Rarely indeed is it necessary for the physician to prescribe more clothing for the newly-born infant, but, on the other hand, he has very frequently to advise the mother to clothe the infant less warmly and less tightly, especially during the hot summer months. As a general principle, the young infant requires warmer clothing than the older child, yet the amount of clothing required must vary with the season. During the winter and cooler months of the year the ordinary flannel abdominal binder may be used for two or three months, and is then to be changed for a knitted band which is held over the shoulders by straps and pinned below to the diaper. Long stockings reaching to the diaper and a short petticoat and dress, suitable in weight and warmth, should be worn. Even in early infancy it is not advisable to have long petticoats and dresses, which have to be folded about the feet and which interfere with the freedom of action of the legs. The feet protected by stockings do not require long swaddling clothes. During the hot months of summer, infants, especially those in our large cities, should be very lightly clad; on very hot days everything may be removed except the light knitted band and diaper.

**Contagion.**—The careful avoidance of contagion is one of the most important principles in the hygiene of infancy. The carelessness with which infants under three months of age, even among the well-to-do classes, are exposed to influenza, bronchitis, and other catarrhal diseases of the respiratory passages is appalling. It is sometimes difficult to convince even intelligent mothers that it is worth while to carefully isolate the young infant from a prevailing house epidemic of la grippe. It is a well-known fact that during the early months of life attacks of coryza, influenza, bronchitis and other contagions, which are little feared by the older members of the family, may readily develop into serious and even fatal pneumonias. A governing principle in every household should be that a sick child should be carefully quarantined from the other children in the family, until the character of its illness is definitely determined, and if this illness proves to be one of the acute infectious diseases the quarantine, so opportunely begun, should be rigidly carried out. Whooping cough, influenza, bronchitis, and pneumonia are dangerous diseases in early infancy, and all of the ordinary infectious diseases of childhood are likely to run a much more severe course in the young infant than they are in the child. The importance, therefore, of having a nursery which may serve as an isolation room for the well infant in the event of contagion in the family is of great importance.

**Excessive Nerve Activity.**—As the child reaches school age excessive nerve activity (the term including brain work and nerve excitement) continues to be a very potent factor in the production of disease. The hygiene of childhood, and especially that pertaining to school life, should therefore protect the growing nervous system of the child, that it may be relieved from all unnecessary strain.[1]

Functional nervous diseases are greatly increased by subjecting the immature nervous systems of young children to the almost constant excitement, nervous strain, and mental activity to which our social order subjects them. To counteract these dangers the teachers and guardians of the young must be taught that the nervous system of the child differs very materially from the nervous system of the adult; they must be convinced that the child, especially in his nervous organization, is *not a little man;* that his nervous system is structurally and functionally immature; that it is excitable, unstable and under feeble inhibitory control; that the sources of reflex irritation in the child are many; that the nerve centers discharge their force more fitfully and more readily than in the adult; that the period corresponding with the onset and establishment of the reproductive function in girls is a time when they are especially predisposed to nervous disease; that the brain of the child is far more receptive, imaginative, emotional, and imitative than that of the adult. They should be

[1] The following paragraphs in this chapter are modified from a series of papers published by the author in the *Archives of Pediatrics* in 1893-94, under the title "Some Physiological Factors of the Neuroses of Childhood," and were subsequently embodied in his monograph on "The Neurotic Disorders of Childhood," E. B. Treat & Co., 1905.

made aware that these and other physiological peculiarities of the nervous system of childhood are made much more potent for evil when they are associated with anemia, malnutrition, and chronic diseases, which interfere with the physical development of the child.

In 1892 W. T. Porter, from an examination of 33,500 boys and girls in the St. Louis public schools, made a most careful study of the "physical basis of precocity and dullness." He demonstrated that children who are advanced in their studies are, on the average, heavier, taller and of larger girth of chest than less advanced children of the same age. If the ability to succeed in school is a measure of mental power, and if successful scholars are, as a rule, better developed physically than the less successful, it follows that mental ability is, on the average, greater in large children than in small children of the same age; in other words, there is in the child a physical basis for precocity.

Porter makes a practical deduction from the law thus established. The entrance to any grade in a school is guarded by examination, and the children found in that grade are such as have passed the entrance examination and have in this way shown their capacity to do the mental labor exacted of them. The greater number of these children are of the same age. The work of this grade is, then, normal for this age, and the average height, weight, and girth of chest of this age form the physical development most often found in children able to do the work of the grade. No child younger than the average age of any grade should be permitted to enter it until a physical examination has shown that his strength will probably be equal to the work, or, as Porter puts it, "No child whose weight is below the average of its age should be permitted to enter a school grade beyond the average of its age, except after such a physical examination as shall make it probable that the child's strength shall be equal to the strain." In determining this, the relation of weight and girth of chest to height is of special importance. Abnormal height is undoubtedly a disadvantage, yet such children may be able to do their school work, provided their physical development is in proportion to their height. If the contrary is the case, the child will be much less able to resist the strain of school life, and should therefore have careful physical supervision and be relieved of school work when he commences to break down under the confinement and mental strain incident to school life.

In protecting children against the ill effects of excessive brain work and nerve excitement, Porter calls attention to the importance of the frequent weighing of growing children, and says that the failure of a child to make the normal gain in weight is no less important a symptom of physical deterioration than persistent loss of weight in the adult. Failure to gain in weight over a period of months should lead, therefore, to an inquiry into the child's physical condition, into his school tasks, into the number of hours he is confined, and into the general hygiene of his home and school life. If this rule is followed many children will be saved from serious nervous breakdowns.

It is my belief that if the various grades in our public schools were guarded by a physical as well as a mental examination, along the lines above indicated, and if persistent loss of weight or failure to gain in weight over a number of months were recognized as reasons for a physical inquiry into the child's capacity to continue in its grade, the functional nervous diseases of childhood would be much less prevalent than they are at the present time. With children of good physical development working within the limitations of their proper grades, there is no danger that a moderate amount of school work will in any way assist in the development of neurotic disease, provided always that the hygienic conditions of the school, especially as to light and ventilation, are good, and provided also that the rules of hygiene pertaining to the home life of the child are carried out as previously outlined. It is especially important that children who spend a great portion of the day in the schoolroom should sleep out of doors, or with wide open windows at night.

The nervous strain and confinement of school life is a very different matter with children of poor physical development, many of whom are unfortunately precocious. The precocity, however, of this type of child is fitful and is not sustained throughout the school year. In every school there is a large number of children who are neurotic, poorly nourished, anemic, and very materially underdeveloped, and not a few of these are suffering from a low grade of glandular tuberculosis. The nervous systems of such children are malnourished, and they are therefore not capable of doing the ordinary work of their grades, and if they are permitted to continue in this work, or if, as is often the case, these children are encouraged to push on into higher grades than the one to which their years and strength would assign them, disastrous consequences will surely follow, and they will become the victims of chorea, hysteria and other neuroses. If the medical supervision of our schools, which at the present time is concentrated upon the prevention of the spread of contagious diseases, could be extended so that the physically weak and malnourished child could be referred to the family physician or other competent medical authority, in order that the question of the advisability of its continuing to do its full school work might be determined before a physical or nervous breakdown necessitated the withdrawal of the child from the school, then school life would be a much less important factor in the production of disease. In dealing with children of poor physical development it is not always advisable to remove them entirely from school; in some instances it may be wise to have the child go to school only during the morning session and to arrange its school work so that it may be accomplished without nervous strain. In other cases the child may be temporarily removed from the school and receive home instruction, which will enable it to keep intellectual pace with the children of its school grade. In every such instance the child must be kept under observation until its physical development fits it for the school grade to which its age and intelligence would assign it.

It is my belief that our public school system should be so remodeled

that all children under twelve or fourteen years of age would be required to spend only the morning or the afternoon at school. The present system, which requires that a child during the winter months should spend nearly all the daylight hours in a schoolroom, is bad. It is a fact which must be evident to every thinking individual that if, instead of spending six or seven hours of the day in a schoolroom filled with other children, where the hygienic conditions are bad and where physical exercise is largely done away with, school children were confined for three or four hours only and were permitted to devote the remainder of the day to outdoor play, their physical condition would be greatly improved and their mental development would not in the least be retarded.

The reasons, then, are clear why we should not allow a child of poor physical development to be pushed to rapid brain development. If we do, its nervous system will surely suffer from the strain, and whatever predisposition it may have to neurotic or other chronic diseases will be greatly increased. In dealing with individual cases it is important that the physician should know the child's hereditary tendencies. He cannot, of course, change the child's ancestry, but he can speak out against the crime of pushing children with hereditary physical defects to rapid brain development, and in doing so he may prevent the development of an hereditary constitutional weakness into an actual disease.

In this demonstration of the injury which results to the nervous system of the delicate child, from the nervous strain and unhygienic conditions of school life, we have a most important warning against the pernicious habit of encouraging mental precocity in early childhood. It is a matter of almost daily experience to see a poorly nourished and perhaps tuberculous child brought forward for the purpose of demonstrating its "wonderful" precocity. The proud mother and overzealous nurse commence the process of mental cramming even before infancy has passed into childhood. From this time on, children are daily being taught, apparently with the idea of destroying their childhood and making of them little men and women. Mothers must be told that *early precocity is an abnormal condition in the human infant,* which, if encouraged, may result in actual disease and permanent mental impairment. They must learn that *vegetation* is the ideal life of infancy and early childhood, and that in order to get the best results they must look to the physical, and retard the intellectual, development of the young child. It must not be taught; it must not be trained; it must have plenty of exercise, fresh air, proper food, and, if possible, should spend a portion of the year in the country away from the clamor and excitement of city life. In the country the older child has more solitude, and must depend more upon his own initiative, the importance of which can scarcely be overestimated in giving independence of thought and character to the future man.

In the modern well-appointed home the child too often has some one to do his thinking, some one to minister to his every want and some one to teach or amuse him throughout his waking hours. He has little time

to himself and a very small portion of his day is spent in play with his intellectual equals. Where these conditions exist there is little chance that the best possibilities in the boy will be utilized for making the best possible man. In 1893 I wrote as follows: "If there is one crying evil common to all of our large cities, it is the scarcity of playgrounds for children, and the attention of humanitarians should be called to this fact. If our generous citizens would pause long enough in the building of hospitals, libraries and places of learning to realize there is a field almost totally neglected by the humanitarian and one of quite as much importance to the welfare of our communities, then possibly a portion of the vast sums of money annually spent in this way would be used in providing playgrounds for children. These playgrounds should not be covered with beautiful grass plots guarded by policemen, but they should be playgrounds in the best sense of the word; places where ball, tennis and all kinds of healthful sport could be enjoyed." Since these words were written much has been done in our large cities to furnish playgrounds for children, and a movement is now apparently spreading over the country, hand in hand with the fresh air movement, which is teaching the poor as well as the rich that healthful play in the open air for a portion of the day and healthful sleep in fresh flowing air at night are much more important to the success of the future man and woman than is the number of hours spent in the schoolroom. The day has apparently dawned when the physiological importance of the physical as well as the mental development of children is to be generally recognized, and cities and philanthropical societies will be called upon to furnish like opportunities for the development of both.

In the hygienic care of young children it is most important that their irritable and undeveloped nervous systems should be protected as much as possible from reflex excitation. The profound nervous disturbances which may be produced by chronic reflex irritation are not fully recognized by those who have the care and teaching of children. Uncorrected eye-strain, adherent prepuce and clitoris, chronic adenoid disease, chronic disease about the rectum and intestinal irritation may be largely responsible for many of the most annoying neurotic disorders of childhood, such as headache, night terrors, incontinence of urine, hysteria, chorea, and general nervous excitability, any one of which may so interfere with the health and comfort of the child as to make it impossible for him to continue in his school work.

The fact that reflex irritation is commonly associated with other factors in the production of disease does not in the least diminish its importance as a cause of neurotic disease. The removal of the reflex excitant in many instances cures the neurosis, even though other important factors remain, and not infrequently our best efforts at removal of other factors of neurotic disease fail to produce a cure as long as the reflex excitant remains to constantly irritate the nerve centers. The explanation of these clinical facts is that reflex irritation does not act simply as an excitant in discharging nerve force from irritable centers, but it also acts in keeping up

the irritability in these centers, and if long continued it produces changes in the nerve centers, recognizable under the microscope. C. F. Hodge has shown that definite changes occur in the nerve cells of the brain and spinal ganglia of certain birds and bees as a result of their normal daily activity. He compared the nerve cells of sparrows and swallows shot in the early morning with the nerve cells of sparrows and swallows shot in the evening after a day of hard flight. Experiments of this kind invariably showed fatigue changes in the nerve cells tired from the day's work. Hodge also found definite changes to occur in the spinal ganglion cells of the frog, the cat and the dog under electrical stimulation, and these changes were very similar to the changes which he had observed to result from the normal daily activity of nerve cells. He also observed that the nerve cell recovered much more slowly than it tired, and concludes that: "Individual nerve cells after electrical excitation recover if allowed to rest for a sufficient time, but the process of recovery is slow. From five hours' stimulation recovery is scarcely complete after twenty-four hours' rest." In these observations we have an explanation of the disastrous consequences which result to the immature nervous system of the child from excessive brain work, nerve excitement, and chronic reflex irritation, and we have also impressed upon us the important physiological fact that these nerve centers, if they are to continue to do their best work and functionate in a normal manner, must have long periods of rest to recover from the fatigue changes which normally result from their physiological activity. It is also a fact that the younger the child the more pronounced will be the fatigue changes resulting from physiological or pathological activity of its nerve cells, and therefore the longer will be the period of rest required to restore these nerve centers to a normal condition.

Reflex irritation, brain work, and nerve excitement are much more potent factors in producing functional nervous diseases in the child than in the adult, for the following reasons:

1. The nervous system of the child is more irritable and unstable by reason of its incomplete functional development.

2. The inhibitory control of higher nerve centers over spinal reflex movements is feebly developed in the child.

3. Blood changes associated with anemia and malnutrition are much more common allies of reflex factors in producing nervous diseases in children than they are in adults.

In the above observations we have not only a physiological but also a morphological explanation of how and why prolonged brain work, nerve excitement and chronic reflex irritation may be such important factors in producing all kinds of neurotic disorders in the young child. It follows, therefore, that in the hygienic supervision of the child, if these diseases are to be avoided, not only all the general hygienic rules which have been outlined in this chapter should be followed, but also that the child should be carefully examined with reference to reflex causes of irritation to the nervous system. It is also important that the physician should recognize

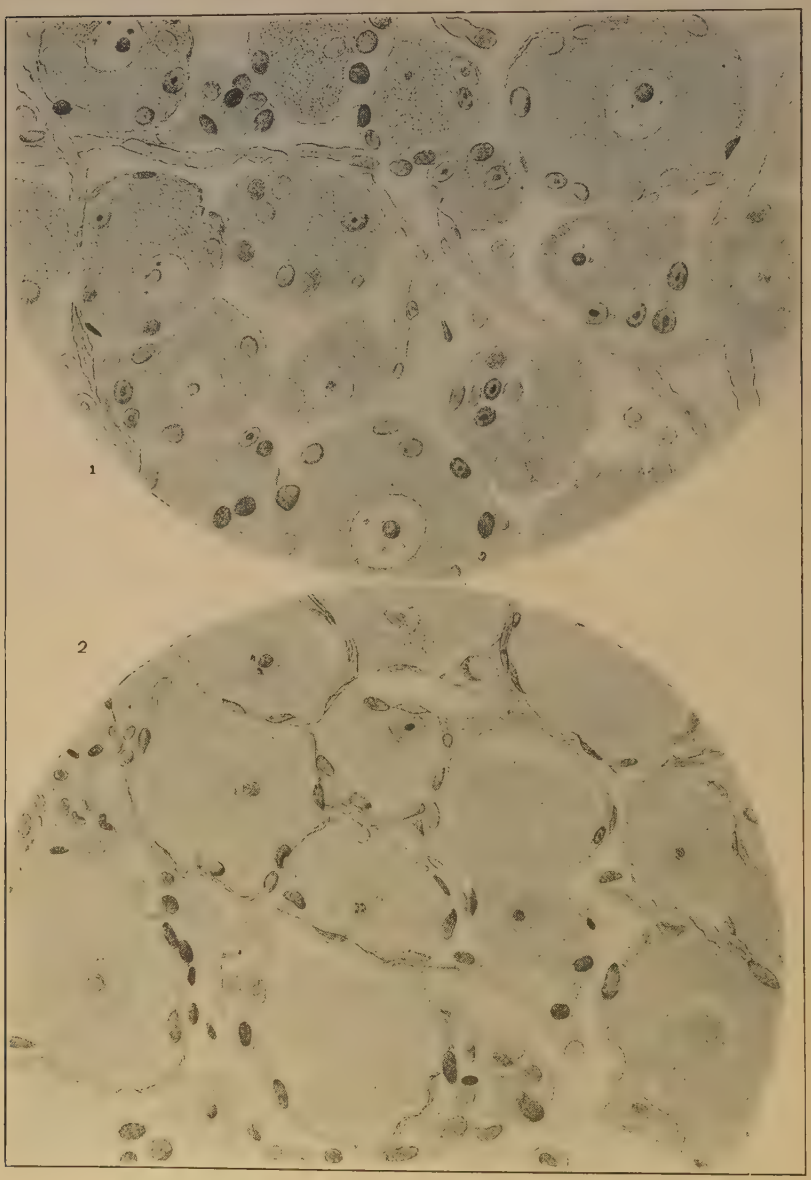



FIG. 1.—ELECTRICAL STIMULATION.—CATS.

1. Normal. Left spinal ganglion of 1st thoracic pair. Osmic acid.

2. Stimulated 5 hrs. Mate ganglion to 1. Osmic acid.

By comparing 2 with 1 is seen the effect of severe work (15 seconds' stimulation to 45 seconds' rest) for 5 hours, the nuclei becoming darker, shrunken and irregular in outline, protoplasm somewhat vacuolated. (C. F. Hodge, *Journal of Morphology*, Vol. VII.)

the fact that the functional development of the male and female genital organs which marks the approach of puberty is a source of marked reflex disturbance which may greatly predispose to neurotic diseases, and that therefore, during this period of life, children should be more carefully guarded from the dangers which may result from excessive brain work and nerve excitement. Above all, the importance of rest to the nervous system should be recognized as the all-important prophylactic measure in preventing disastrous results from the above named causes of disease, and that this rest can only be satisfactorily obtained by prolonged periods of sleep. Sleep is "nature's sweet restorer," and its importance as a preventive of disease in childhood cannot be overestimated. It is a most important part of the hygiene of infancy and childhood that children from the beginning of their lives should, by regulating their daily routine and by placing them under quiet surroundings, be made to sleep as much as possible. Even after the child has reached the school age it should be sent to bed very soon after its evening meal so that if possible it may have eleven or twelve hours of sleep. If the habit of long and undisturbed sleep is engrafted firmly upon the infantile nervous system, it is, as a rule, easy to continue it into late childhood, and if parents could only realize the enormous benefit, physical and mental, which its continuance would bring to the child, then they would guard and protect the sleeping habit as one of the most important heritages of infancy.

## CHAPTER II

### GROWTH AND DEVELOPMENT

**Weight during Infancy and Early Childhood.**—The increasing weight of the infant and young child along normal lines is by far the best indication of satisfactory growth and development. There are many signs and symptoms which tell of the unsatisfactory development of the child, but these for the most part derive their importance from their association with a failure to gain in weight, or an actual loss in weight. For example, an infant that is making the normal gain in weight week after week on breast milk may have curds or occasional mucus in its stools, or the discharges from the bowel may vary in consistency, may at times be green in color, and the infant may from time to time suffer with attacks of colic, and yet all or any of these symptoms pointing to intestinal indigestion are, as compared with a normal increase in weight, of comparatively little importance. In such cases, of course, efforts should be made to correct the indigestion of the infant by regulating the life and diet of the mother or by other means outlined in the chapter on Infant Feeding; but the fact that there is a steady and normal gain in weight, notwithstanding the other symptoms that may be present, is of itself sufficient reason for continuing the child upon the mother's milk. On the other hand, the stools may be normal and

the infant may be comparatively contented with its food, sleeping and behaving in a normal way in all other particulars except that it is failing to gain in weight, and this last indication, outweighing all of the others, indicates that the food of the infant must be supplemented or changed.

Failure to gain in weight in infancy and childhood has the same pathological significance as loss of weight in the adult. It is not enough that the infant or young child should hold its own in weight; it should, if its nutritional problems are properly solved, increase in weight in the ratio that is normal for its age. Slight variations in weight occur from unknown causes in the young infant; the weight may remain stationary for three or four days at a time or the scales may record, within a day or two, a gain of three or four ounces. These slight variations in weight from day to day should be entirely disregarded except perhaps in very ill or in very young or premature infants. The child, under ordinary conditions of development, should be weighed but once a week during the first seven months of life, and thereafter but twice a month during the first year. During the second year of life it should be weighed once a month, and during the third and fourth years at intervals of every three or four months. By these regular weighings, which are especially important during the first year, very valuable information is obtained as to the growth and development of the child. Failure to gain in weight for one or even two weeks may not always be an indication that the infant is not getting proper food in sufficient quantities, since not infrequently the same infant after such a standstill may on the same food commence to gain in weight. Too much importance, therefore, must not be placed upon the temporary failure to gain in weight of an infant that has previously been developing along normal lines. If the failure to increase in weight, however, continues longer than three weeks, and especially if this symptom be associated with others indicating that the child has insufficient or improper food, then prompt steps should be taken to find the cause of the trouble and to relieve it. On the other hand, it is well to remember that infants, especially those fed upon the patent foods, may increase rapidly in weight and yet not be properly nourished. The condensed milk and patent food babies, while they are increasing rapidly in weight, may be suffering very seriously in the development of their osseous, muscular and nervous systems; increase in weight on ill-balanced foods may go hand in hand with the development of rickets. While it is true that the increasing weight of the infant is the most important indication of its satisfactory growth and development, it is also true that, if one depends alone upon this sign of normal development, many unfortunate mistakes will be made. The increase in weight as a sign of growth and development is to be studied in connection with other evidences of good health or disease. Weight observations are made, as a rule, by the mother or nurse, and too often are they taught to rely exclusively upon increase in weight in determining the physical condition of the infant. All infants, even those that are satisfactorily gaining in weight, should be seen and carefully examined from time to time by the physician

in order to determine their true physical condition. The infant should be stripped and weighed upon the same scales, by the same person and at the same time of day, so that all the conditions may be as nearly alike as possible at the different weighings.

The average birth weight of the normal infant at term is about seven pounds; female infants are, on the average, from one-half to one pound lighter than males. Great variations in the birth weight may occur; in full-term infants which on inspection appear to be satisfactorily developed a birth weight of over six pounds may be considered normal. In proportion as the birth weight falls below six pounds the vitality of the infant is impaired and its chances for normal development diminished. The infant loses in weight for two or three days following its birth; the most rapid loss occurs during the first day, and by the third or fourth day the child begins to slowly gain in weight. This initial loss of weight amounts to from five to seven ounces; the lowest weight of the baby is commonly found on the third day, and by the end of the first week it regains its birth weight. This loss of weight is largely due to the discharge of meconium and urine and the absence of food and water. If newly-born infants are given water to drink (which may be fed to them with a medicine dropper, two or three teaspoonfuls every three hours), the initial loss of weight here described will be diminished, and the water thus given will serve a valuable purpose in increasing the urinary secretion and washing out the tubules of the kidney.

From the end of the first week the normal infant gains rapidly in weight, beginning with an ounce a day, or about two pounds every month. This rate of increase is maintained up to and perhaps throughout the third month of life. Toward the end of the third month and throughout the fourth there is a slight falling off in this gain of weight; during this time the increase is gradually diminished to five ounces a week, and the infant still continues to gain slightly less in weight, until at the end of the sixth month it is gaining only four ounces a week. From this time to the end of the year it averages from three to three and one-half ounces a week. By this rate of increase an infant weighing seven pounds at birth should weigh fourteen pounds at six months and twenty pounds at one year of age. This wonderful growth, by which the infant doubles its weight during the first six months of life, and almost triples its weight by the end of the first year of life, together with the rapid heat loss of this period, is responsible for many of the physiological and pathological peculiarities of infancy, and explains the large amount of food required per kilogram of weight, the intense activity of the metabolic processes, and the great demands made upon the excretory organs during this period of life.

After the first year of life the increase in weight gradually becomes less rapid. During the second year the child gains about three-fourths of a pound a month, or nine pounds during the year. In the third year of life it gains about four and one-half pounds; during the fourth year its weight is increased three pounds, and in the fifth year two and one-half pounds, at which time it should weigh from forty to forty-one pounds. From the end

of the first year to the end of the fifth year of life the weight of boys is from one to one and one-half pounds heavier than girls, and this superiority of weight in boys is maintained for a number of years with little variation. The acceleration in weight, however, which precedes puberty takes place earlier in girls than it does in boys, and for a time during this period of their lives the girls are the heavier. A little later the boys regain their superiority in weight and retain it until maturity. The actual and comparative rate of growth of 33,500 boys and girls in the public schools of

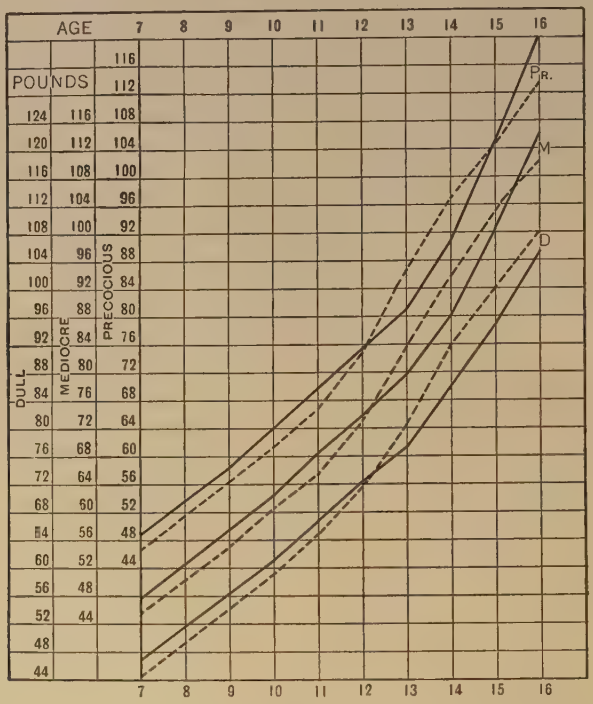



Fig. 2.—The Rate of Growth in Weight of Dull, Mediocre and Precocious Boys and Girls.

Full lines represent boys' weight. Dashed lines represent girls' weight. (W. T. Porter.)

St. Louis is graphically shown in Figure 2, taken from W. T. Porter. He says: "The growth of boys and girls runs a parallel course in early childhood. At age six boys are heavier than girls, and this advantage is maintained for several years. But when the difference of sex begins to make itself felt, the relation between the weight of the sexes is changed, the boys lose their superiority and the curve of girls' weight rises above theirs. This difference persists for about three years, and then the curves once more cross and the youth is once more heavier than the maid. In the plate the curves of girls' weight cross the boys' curves at the same age in dull, mediocre and precocious children."

**Height of Child at Different Ages.**—The average length of the male newly-born infant is twenty inches, the length of the female is about one-half inch less. According to Rotch, whose figures are in close accord with those of other observers, the most rapid growth occurs within the first month, during which time the infant is increased in length one and three-fourths inches and during the second month one and one-half inches. From the third to the twelfth month the rapidity of growth becomes gradually less, until at the end of the first year of life the average increase is about one-half inch per month. The child gains eight inches in length during the first year; three and one-half inches during the second year, and three inches during the third year; thereafter the gain is from two to two and one-half inches each year up to the eleventh year of life. As the period of puberty approaches there is a more rapid gain in height, which corresponds rather closely with the rapid increase in weight which occurs at this period. This increased rate of growth begins about the twelfth year in girls and about the thirteenth year in boys and continues for two or three years, and during this time the increase in height is from three to four inches per annum. It is not improbable that the rapid body growth and rapid functional development of the nervous system, which are frequently associated with nervous irritability, mental precocity, tachycardia, headache, and other nervous symptoms, may be produced by excessive activity of the thyroid gland which occurs with the approach of puberty. At any rate, it is important to recognize the fact that during this period of rapid growth and development the child is to be carefully protected from excessive brain work and nerve excitement.

The following tables from Koplik give the average height, weight, head circumference and chest measurements of American boys and girls. They are collated from thousands of children in various states by Bowditch, Burk, MacDonald, Hastings and Chapin:

TABLE 1

*From Birth to Four Years of Age*

| Age | Sex | Length | | Weight | | Head Circum. | | Chest Girth | |
|---|---|---|---|---|---|---|---|---|---|
| | | In. | Cm. | Lbs. | Kilos. | In. | Cm. | In. | Cm. |
| Birth..... | Boys.... | 19.7 | 50.0 | 7.4 | 3.45 | 13.8 | 35.1 | 12.6 | 32.0 |
| | Girls.... | 19.3 | 49.0 | 7.1 | ..... | 13.1 | 33.4 | 11.8 | 30.0 |
| 6 months... | Boys.... | 25.4 | 64.8 | 16.0 | 7.2 | 16.0 | 40.5 | 15.7 | 39.9 |
| | Girls.... | 25.0 | 63.6 | 15.5 | 7.0 | 16.4 | 41.7 | 15.2 | 38.6 |
| 12 months... | Boys.... | 29.5 | 73.8 | 21.5 | 9.8 | 17.8 | 45.3 | 17.8 | 45.1 |
| | Girls.... | 28.7 | 73.2 | 21.0 | 9.5 | 18.2 | 46.3 | 19.0 | 48.3 |
| 2 years..... | Boys.... | 33.8 | 84.5 | 30.3 | 13.8 | 19.3 | 49.0 | 20.0 | 50.8 |
| | Girls.... | 32.9 | 82.8 | 29.2 | 13.3 | 18.0 | 45.6 | 18.0 | 48.0 |
| 3 years..... | Boys.... | 37.0 | 92.6 | 34.9 | 15.9 | 19.3 | 49.0 | 20.1 | 51.1 |
| | Girls.... | 36.3 | 90.7 | 33.1 | 15.0 | 19.0 | 48.4 | 19.8 | 50.5 |
| 4 years..... | Boys.... | 39.3 | 98.2 | 37.9 | 17.2 | 19.7 | 50.3 | 20.7 | 52.8 |
| | Girls.... | 38.8 | 97.0 | 36.3 | 16.5 | 19.5 | 49.6 | 20.5 | 52.2 |

TABLE 2

*From Five and a Half to Fifteen and a Half Years*

| Years of age | Sex | Height | | Weight | | Head Circum. | | Depth of Chest | | Breadth of Chest | | Chest Expansion | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | In. | Cm. | Lbs. | Kilos. | In. | Cm. | In. | Cm. | In. | Cm. | In. | Cm. |
| 5½... | Boys.... | 41.7 | 105.9 | 41.6 | 18.9 | 20.1 | 51.2 | 4.9 | 12.3 | 7.1 | 18.1 | 1.3 | 3.4 |
| | Girls.... | 41.3 | 104.9 | 40.7 | 18.5 | 19.7 | 50.2 | 4.8 | 12.3 | 7.0 | 17.7 | 1.4 | 3.5 |
| 6½... | Boys.... | 43.9 | 111.9 | 45.2 | 20.5 | 20.2 | 51.5 | 5.0 | 12.8 | 7.2 | 18.4 | 1.6 | 4.2 |
| | Girls.... | 43.3 | 109.0 | 43.4 | 19.5 | 19.8 | 50.3 | 4.9 | 12.3 | 7.0 | 17.7 | 1.5 | 3.8 |
| 7½... | Boys.... | 46.0 | 116.8 | 49.5 | 22.5 | 20.4 | 51.9 | 5.1 | 12.9 | 7.4 | 18.9 | 1.8 | 4.5 |
| | Girls.... | 45.7 | 116.0 | 47.7 | 21.6 | 20.0 | 50.9 | 4.9 | 12.5 | 7.2 | 18.4 | 1.8 | 4.5 |
| 8½ . | Boys.... | 48.8 | 123.9 | 54.5 | 24.4 | 20.5 | 52.2 | 5.1 | 12.8 | 7.6 | 19.4 | 2.3 | 5.9 |
| | Girls.... | 47.7 | 121.1 | 52.5 | 23.8 | 20.2 | 51.2 | 4.9 | 12.5 | 7.4 | 18.9 | 2.0 | 5.0 |
| 9½ . | Boys.... | 50.0 | 127.0 | 59.6 | 27.0 | 20.6 | 52.4 | 5.2 | 13.2 | 7.8 | 19.7 | 2.5 | 6.5 |
| | Girls.... | 49.7 | 126.2 | 57.4 | 26.0 | 20.4 | 51.9 | 5.1 | 13.1 | 7.0 | 19.3 | 2.2 | 5.6 |
| 10½ . | Boys.... | 51.9 | 131.8 | 65.4 | 29.5 | 20.6 | 52.6 | 5.2 | 13.2 | 8.0 | 20.2 | 2.7 | 7.0 |
| | Girls.... | 51.7 | 131.3 | 62.9 | 28.5 | 20.5 | 52.0 | 5.1 | 13.0 | 7.8 | 19.8 | 2.4 | 6.0 |
| 11½ . | Boys.... | 53.6 | 136.1 | 70.7 | 32.2 | 20.8 | 52.9 | 5.4 | 13.8 | 8.2 | 20.9 | 2.9 | 7.3 |
| | Girls.... | 53.8 | 136.6 | 69.5 | 31.5 | 20.7 | 52.5 | 5.2 | 13.1 | 8.0 | 20.3 | 2.6 | 6.6 |
| 12½ . | Boys.... | 55.4 | 140.7 | 76.9 | 34.9 | 21.0 | 53.3 | 5.6 | 14.1 | 8.5 | 21.5 | 3.0 | 7.8 |
| | Girls.... | 56.1 | 142.5 | 78.7 | 35.7 | 20.9 | 53.0 | 5.4 | 13.8 | 8.4 | 21.0 | 2.4 | 6.2 |
| 13½ . | Boys.... | 57.5 | 146.0 | 84.7 | 38.5 | 21.1 | 53.5 | 5.6 | 14.3 | 8.7 | 22.7 | 3.2 | 8.2 |
| | Girls.... | 58.5 | 148.6 | 88.7 | 40.3 | 21.0 | 53.5 | 5.5 | 14.1 | 8.7 | 22.1 | 2.6 | 6.6 |
| 14½ . | Boys.... | 60.0 | 152.3 | 95.2 | 43.2 | 21.3 | 54.1 | 5.9 | 15.0 | 8.9 | 22.7 | 3.3 | 8.4 |
| | Girls.... | 60.4 | 153.4 | 98.3 | 44.6 | 21.3 | 54.1 | 5.7 | 14.5 | 9.0 | 22.9 | 2.7 | 6.8 |
| 15½ . | Boys.... | 62.9 | 159.7 | 107.4 | 48.8 | 21.4 | 54.5 | 6.3 | 16.0 | 9.3 | 23.6 | 3.3 | 8.4 |
| | Girls.... | 61.6 | 156.4 | 106.7 | 48.5 | 21.5 | 54.6 | 6.0 | 15.3 | 9.5 | 23.8 | 2.6 | 6.5 |

**Head Measurements.**—Slight variations from the head measurements in the above tables recording the maximum circumference at different ages of infancy and childhood may occur without special pathological import, but marked variations usually have pathological significance. On the average the mentally defective have smaller and less symmetrical heads than normal children of like age. When symptoms indicating imbecility in the infant exist, a small circumference of the head associated with lack of symmetry of the skull would be confirmatory evidence. An unusually large circumference of the head when associated with other signs of hydrocephalus may also point to lack of mental development. It should also be remembered that a comparatively large head is a not uncommon symptom of rickets and cretinism.

The anterior fontanels, even in the normal infant, may vary in size from one-half inch in both its diameters to a lateral measurement of two and one-half inches, and an anteroposterior measurement of three inches. This opening may not materially decrease in size until the eighth or ninth month, when it gradually begins to grow smaller. At the end of the year it should not be more than one and one-half inches in diameter and should be closed by the eighteenth or twentieth month. Variations as to the time of closure of this fontanel may occur, within the limits of good health, from the end of the first to the end of the second year of life. Its failure to close by the end of the second year of life is commonly an indication of rickets, of malnutrition, or of some more serious disease such as hydrocephalus or imbecility. The posterior fontanel commonly closes within the first six weeks, but in deciding upon the pathological import of open fontanels, after their normal period of closure, other symptoms must be taken into con-

sideration. In microcephalic skulls the fontanels may close and premature ossification of the sutures may occur early. This condition is associated with a small, asymmetrical head and lack of development of the brain. Both hydrocephalus and microcephalus are elsewhere described. The softness of the skull bones and the open fontanels of the young infant predispose it to pressure deformities. At this age permanent deformities or irregularities in the shape of the skull may be produced by permitting the infant to habitually rest its head in one position.

**Development of the Spine and Bony Framework.**—The spine of the infant at birth, as Rotch has noted, contains so much cartilage and so little bone, and is so feebly supported by weak and undeveloped ligaments and muscles, that it can be easily bent, twisted, and deformed. Instead of the normal curves of the self-sustaining spinal column of late childhood and adult life, there are present the position curves which result from bending and twisting the soft flexible spine of the infant under the influence of weight and pressure. The young infant should spend nearly all of its time in a prone position. It should, however, be shifted from side to side, or back to stomach, so that habitually lying in one position may not predispose to permanent pressure deformity of the head or the spine. As the infant grows older, the spine becomes stronger and more capable of assisting in the support of the body of the infant, but it is important that during the latter half of the first year of life the infant be not encouraged to sit up too frequently or too long at a time. Throughout early childhood there is a gradual development in the bones and supporting ligaments and muscles of the spine, but long after school life has begun the spinal column still remains so flexible that permanent deformities may result from wrong positions in writing or other work over low school desks, which cause the child to sit for hours each day with a bent or twisted spine. This is a common cause of spinal curvatures in school children, many of which are so pronounced that the most skilful orthopedic treatment over a period of years fails to remove the deformity entirely.

The whole bony framework of the child is in an active stage of growth and functional development, but from a pathological standpoint the most important processes are taking place in the epiphyses of the long bones, and in the small bones which enter into the formation of the ankle and wrist. This rapid metabolism and growth of new bone predispose this part of the bony framework to tuberculous, pyogenic and other infections.

Pryor used the development of the bony framework of the wrist as an anatomical index to the general development of the skeleton, and his tables show that the bony development is more advanced in girls than it is in boys of the same age. Rotch in a very clever and far-reaching research has demonstrated that Röntgenographs of the carpal bones and epiphyses of the radius and ulna may be used to indicate the actual bony and muscular development of the child. In this way may be determined the anatomic age, which does not always correspond to the chronologic age of the child. The anatomic age, as determined by the degree of development of the

carpal bones and epiphyses of the radius and ulna, determines the degree of development of the whole bony and muscular framework of the child, and is a fairly accurate index of its capacity for mental and physical work. Rotch says, "The people must be educated up to the plane of intelligently seeing that, because an individual has been born three or four years, this does not necessarily mean that such chronologic age should be rigidly adopted for entering a kindergarten; that, because it is six or eight years of chronologic age, it should necessarily be in the usual grade in school corresponding to that age; that, because it is ten or twelve chronologic years of age, it should necessarily be grouped in athletics with boys or girls of that chronologic age; or, because it is fourteen, fifteen or sixteen years of age, it should be allowed to work beyond what its anatomic development shows it can do without physical harm, as, for instance, in the mills."

FIG. 3a.—BAD POSITION WHILE WRITING.
(After Hoffa in Pfaundler and Schlossmann.)

Rotch has worked out a practical system of grading children for school and other work which will correct, he believes, the errors which result from the classification of children for this work by their chronologic age, or by their apparent physical development as determined by weight and height. The value, however, of the weight and general appearance of the child as a simple and practical method of classifying him for his school work has been already referred to and must for a time at least remain the simplest and most practical method of classification. The more scientific and more accurate method of determining the anatomic age of the child by Röntgenization may, as Rotch believes, in time supersede the cruder and simpler methods.

**Muscular Development.**—The general bony framework of the body keeps pace with the muscular development of the child and attains sufficient stability to support the positions of the body which the child instinctively assumes in the physiological activity of its developing muscles. It is inadvisable, however, and may be positively injurious to place the child in

sitting or standing positions before its muscular development is such that it voluntarily or instinctively attempts to assume these positions. The young infant as it lies upon its back should have perfect freedom of muscular movement, and as its muscular development grows apace it instinctively attempts to change its position. It does not have to be taught to turn over in bed, to lift its head from the pillow, to attempt to crawl or to make an effort at sitting up or climbing upon its feet; all of these movements are instinctive in the normal child, and they are exercised as soon as its muscular development is equal to the physical effort which these movements entail, and as soon as the bony framework of the body is able to assist, without injury, in supporting the infant in these positions. At birth the muscular development of the hands and forearms is relatively stronger than the rest of the body. The young infant clings to objects that are placed in its hand, and in the latter part of the third or beginning of the fourth month it reaches for and takes hold of objects which are placed in front of it. At birth the infant is unable to hold its head in an upright position, but during the third or fourth month the muscles of the neck have sufficiently developed for the child to lift its head from the pillow and hold it in an upright position for a short time. From this time on the infant begins to assume positions in which the whole spinal column is held in a mild degree of temporary rigidity. By the eighth or ninth month it may be able to sit alone for a short time, and about this time it begins to crawl and attains in a mild degree the faculty of voluntary locomotion. About the ninth or tenth month its first attempts at pulling itself upon its feet are made, and thereafter it soon acquires the power to stand and to make an effort at walking, when it is supported by a chair or some other object. When eleven or twelve months of age it may, while clinging to some object, begin to walk, but is not, as a rule, able to walk alone until it is fifteen or sixteen months of age. It should be remembered,

Fig. 3b.—Bad Position While Writing.
(After Hoffa in Pfaundler and Schlossmann.)

however, that even in normal infants there is considerable variation as to the time when they begin to make various movements. Some may walk alone when ten or eleven months of age, and others without apparent evidence of disease may not attain this faculty until they are eighteen months of age. The late development, however, of the physical functions here outlined is, especially when associated with other symptoms, important evidence of disease or lack of development on the part of the nervous system. Inability to hold the head upright at the fifth month, failure to reach for and grasp objects in the fifth or sixth month of life, failure to maintain a sitting position at one year of age, no inclination to assume the upright position at the eighteenth month and failure to walk when two and one-half years of age are indications not only of lack of muscular and bony development but of nervous development as well. In such instances a careful search should be made for other signs of disease or lack of development of the nervous system.

**Special Senses.**—The infant during the first few days of life is deaf and this condition perhaps continues until the Eustachian tubes are cleared of mucus, and air has found its way into the internal ear. During the first three or four days the infant, for this reason, sleeps soundly, undisturbed by surrounding noises. From the third to the fifth day there are indications that the child hears and thereafter this sense gradually becomes more acute until by the end of the first month of life the child is disturbed by slight noises, and by the end of the fifth or sixth month it is able to distinguish between and interpret certain noises such as the sound of individual voices. After this time infants are very sensitive to and easily frightened by loud and unaccustomed noises.

Soon after birth the eyes are sensitive to light and for this reason during the first weeks of life they should not be exposed to bright lights. At the end of the second or third week the eyes of the infant will follow a bright light as it is moved in front of its face. From this time on it notices more and more bright moving objects, and at the end of the third month it not only sees all objects within its range of vision, but it may give evidence that it recognizes its nursing bottle or some other object which it has seen before.

The sense of taste is perhaps slightly developed at birth, as an infant a few days old takes sweetened food more readily than it will sour or bitter liquids. This inborn preference for sweet food is not to be unduly encouraged, as even very early in the life of the infant it may lead, in artificially fed infants, to the giving of an excess of sugar simply because the infant demands it.

The function of speech is developed earlier in girls than it is in boys. At the twelfth or thirteenth month the infant may say mama and papa, toward the close of the second year simple short sentences may be used, and thereafter the faculty of speech is more rapidly developed. It is not a very unusual thing in children of normal intellectual development to have the speech faculty delayed for a year or more after the time when it is

usually acquired by the normal child. These children all through the latter half of the first year of life and through the second year manifest the same signs of intelligence as the normal child; they understand everything that is said to them and make their wants known by signs and by articulate sounds which by long use have become intelligible to those around them.

**Nervous System.**—The dura mater in the infant is closely adherent to the skull, and the blood vessels of the pia mater are so abundant and so fragile that hemorrhage into the subarachnoid space may result from causes which produce high blood pressure. This accounts for the not infrequent occurrence of cortical hemorrhage during labor and also explains why paroxysms of whooping cough and severe convulsions may produce this accident in older children. The serious effect on the mental and physical development of the child which results from these hemorrhages is not so much due to their extent as it is to the fact that they produce a permanent cortical lesion in the young and immature brain which interferes with its structural and functional development.

Of all the vital organs the nervous system at birth is both structurally and functionally the most immature. Throughout infancy and childhood the nervous system develops very rapidly in size and structure and much less rapidly in function. In the later years of childhood the functional development of the entire nervous system is much more rapid. From birth up to the seventh year of life the brain develops enormously in weight, in structure, and much less rapidly in function. At this time it has attained 90 per cent. of its maximum weight (Boyd), and thereafter it slowly increases until it has attained its maximum size at the age of eighteen; but increase of function does not keep pace with increase of weight. While the brain of the child at eight is almost as large as that of the adult, as Clouston says, "The difference between what the brain of a child of eight and the brain of a man of twenty-five can do and can resist is quite indescribable. The organ at these two periods might belong to two different species of animals so far as its essential qualities go." The important structural changes in the brain of the child pertain to the development of the convolutions and their arrangement in groups which preside over special functions, but it should be remembered that even in the normal child irregularities may occur in the order of development of both structure and function. It is quite within the limits of health that certain functions of the nervous system may be rapidly developed and that others may be unusually retarded. While this is true, the important pathological fact must be kept in mind, that many factors such as heredity, nutritional conditions and environment have a very powerful influence on both the structural and functional development of the nervous system, and these factors, if unfavorable, seriously retard the growth of the nervous system in both structure and function and very commonly interfere with the order of development of important functions.

The most important clinical fact to be derived from these observations

is that the nervous system of the infant and young child, by reason of its rapid growth and immaturity, is characterized by marked nervous irritability and extreme excitability, and that this normal irritability and excitability of nerve cells and nerve centers in the young nervous system may be greatly exaggerated by malnutrition, bad heredity and unfavorable environment. The above facts in part explain the peculiar susceptibility of the child to functional nervous diseases and also explain the powerful influence of bad heredity, malnutrition and unfavorable environment in developing and aggravating these disorders at this period of life.

Feeble inhibition is one of the most important peculiarities of the nervous system of the child. The inhibitory mechanisms which control the discharge of nerve force that regulates such vital processes as the action of the heart and lungs are fairly well developed at birth, while those that regulate reflex phenomena are very immature, and are slowly developed throughout infancy and early childhood. The late development of the function of inhibition is a fact of great importance from a clinical standpoint, because this is the last function of the cell to be developed, and is therefore the one that is most likely to be still further retarded in development by bad heredity, malnutrition and unfavorable environment. *It is the abnormally feeble inhibition which occurs in the abnormal child, brought about by the above-named unfavorable conditions, that is such a powerful factor in the production of neurotic disease in infancy and childhood.*

One can readily understand how feeble inhibition even in the normal child, by reason of an insufficient control over the convulsive centers at the base of the brain and reflex centers in the spinal cord, may predispose to convulsive and other neurotic disorders of childhood, and one can also readily see how bad heredity, malnutrition and unfavorable environment, by retarding the normal development of the inhibitory function of the nervous system, may be all-important factors in the development of these neuroses.

Certain of the reflex centers in the spinal cord which preside over special functions, such as urination and defecation, are so functionally immature at birth that there is a lack of tone of the sphincter muscles over which they preside, and as a result we have an incontinence of feces and of urine. These reflex centers are slowly developed until, under their control, the muscles of the bladder and rectum acquire the normal muscular tone which fits them for the purposes they are to serve, and with this development the centers in the cord assume control of the rectum at about the fifth month and complete control of the bladder muscles at about the end of the second year of life.

It is a fact of great physiological and pathological importance that in the development of the spinal cord the fibers of the pyramidal tract are the latest to become myelinated. At birth they have almost no myelin sheaths and until these are developed it is believed that motor impulses cannot readily be carried from the brain to the spinal cord cells. [1] "Day by

[1] For a fuller discussion of the physiological peculiarities of the nervous system during infancy and childhood, see the author's monograph "Neurotic Disorders of

day as these myelin sheaths are developed the cerebral and spinal motor cells are brought into closer communication so that at about the third or fourth month this communication may be said to be fairly well established; prior to this time the communications are imperfect." These physiological facts may be offered in explanation of the comparative immunity which young infants have from convulsive disorders during the first few months of their lives, and they may also explain the development of spastic palsies as late as the third or fourth month of life which are due to natal and prenatal injuries to the brain.

**Development of the Heat-regulating Mechanism.**—The rectal temperature of the normal newly-born infant at term ranges between 99.5° and 100.5° F., and this temperature is maintained with little variation throughout the first nine months of life. During the second year of life the normal temperature varies from 99° to 100° F., and thereafter throughout early childhood the rectal temperature is from 99.6° to 100.5° F. Throughout infancy and early childhood variations in temperature between 98.5° and 100° F. are of little or no pathological significance. Under the heading, Management of Premature Infants, the fact is noted that in congenitally weak infants the temperature ranges much lower than the figures here given. It is a notable fact that slight causes acting upon the unstable heat-regulating mechanism of the infant and young child will produce high and variable temperatures, while the same causes acting upon the mature nerve centers of the adult may produce no variations in the temperature curve.

The thermogenic or heat-producing centers are located at the base of the brain. These centers have the function of discharging force which will increase tissue metabolism and thereby increase the body heat. Any disease or injury which destroys the efficiency of these centers would cause a decrease of the body heat, and any condition which increases the irritation of or unduly excites these centers would increase the body heat. Before birth the thermogenic centers are in a state of immature functional development. In the human infant born prematurely they are so imperfect that artificial heat is necessary to keep the body warm, and this artificial heat must continue to be supplied until these immature centers have developed to such a state of physiological competency that they are able to supply to the body the normal amount of heat. In the normal infant at birth, although these centers have a fair degree of physiological competency, they are still immature, and much more unstable than they are in the finished brain of the adult. The comparative instability and excitability of the thermogenic centers of the infant and young child cause them to discharge their nerve force and increase the body temperature from slight causes, and in this fact we find one of the explanations of the proneness of infants and young children to develop high temperatures from slight causes.

Like other nerve centers in the unfinished brain of the child the thermo-

Childhood,'' 1905, and ''Clinical Significance of Lack of Development of the Pyramidal Tracts in Early Infancy,'' *Archives of Pediatrics*, 1910.

genic heat centers have their normal irritability and excitability very greatly exaggerated by neurotic inheritance, malnutrition and unfavorable environment. It is the abnormal excitability of these centers in the nervous, malnourished, anemic child that explains the special predisposition which some children have to fever from slight causes.

The thermoinhibitory centers are located in the cerebral cortex and it is their function to control or prevent the discharge of nerve force from the thermogenic centers. The thermogenic and thermoinhibitory centers have their functions so nicely balanced in the normal adult nervous mechanism that with the aid of the heat-dissipating centers they are able to maintain the body at almost a uniform temperature under the most adverse circumstances. But in the infant and young child the thermogenic centers are not only irritable and excitable, but they are under comparatively feeble inhibitory control from the thermoinhibitory centers, and this feeble inhibition of the thermogenic centers, which predisposes the normal child to high and variable temperatures from slight causes, is much more feeble and therefore much less effective in children of bad heredity, malnutrition, and unfavorable environment. It is therefore the abnormally feeble inhibition of the nervous, malnourished, anemic child which leads to loss of control of the thermogenic centers, that especially predisposes certain children to high and variable temperatures. McAllister says, "The inhibitory is the first portion of the heat-regulating mechanism to fail under injury or disease."

The heat-dissipating mechanism plays a much more important part in regulating the temperature of the body in the infant and child than it does in the adult. This is the mechanism by which the infant keeps itself cool when from insufficiency or maladjustment of the thermogenic and thermoinhibitory centers the temperature of the child is raised above the normal point. The dissipation of heat by radiation and conduction and by the constant evaporation of water from the surface of the body is much more rapid in the infant, because the area of skin surface is from four to six times greater in the infant and young child in proportion to its body weight than it is in the adult. For these reasons the heat-dissipating mechanism of the infant is four times as effective as it is in the adult. In the above physiological facts we have an explanation of why the high temperatures of infancy and childhood are so readily reduced by the heat-dissipating mechanism, and we also have an explanation of the comparatively rapid reduction of temperatures at this time of life from hydrotherapeutic measures and from medical antipyretics. These latter act not only on the thermogenic centers in diminishing the amount of heat produced, but they also act through the vasomotor nervous mechanism and cause a profuse perspiration with a resulting rapid evaporation of the water from the surface of the body. It will thus be seen that the heat-dissipating mechanism in infancy and childhood is much more effective than it is in later life, and that in the play of function between this mechanism and that of the heat-generating and heat-inhibiting mechanism we have an

explanation, not only for the high temperatures which occur during infancy and childhood from slight causes, but also for the unusual variations in the temperature which occur at this period of life. As the infant develops into the child and as the child grows older there is a gradual functional development of the heat-regulating apparatus. The thermogenic centers become less irritable and are therefore not so easily excited to produce fever from slight causes. The thermoinhibitory centers gradually increase their efficiency and exercise more and more control over the thermogenic centers; this is especially important in increasing the stability of these heat centers, and of preventing fever from slight causes. With this increase in stability and functional capacity of the heat centers, the heat-dissipating mechanism also becomes more stable and does not respond so readily to hydrotherapeutic and other measures commonly used for the reduction of temperature.

**Evaporation of Water from the Air Passages.**[1]—In certain animals, the dog for instance, which do not sweat, the evaporation of water from the air passages is the chief means of reducing the body temperature. Richet calls the rapid respirations of the panting dog *polypnea;* by these rapid respirations, amounting to as many as four hundred in a minute, the heat of the body is rapidly given off. Richet proved that the polypneic center was not affected by the amount of carbonic acid or oxygen in the blood, and that it was solely for the purpose of heat dissipation.

Does the polypneic center exist and is it functionally active in infancy and childhood? The answer to this question has important clinical bearings. Ott says: "In infants we see a polypnea during fever, the respiration rises in frequency with the rise in temperature." Every physician must have seen many cases of rapid respiration in children that could not be accounted for by pulmonary disease. It not infrequently happens that a child with fever will have sixty, eighty and one hundred respirations per minute, without presenting any sign or symptom of lung trouble. Polypnea is, to my mind, the explanation of this phenomenon. Very rapid breathing is a common symptom of gastrointestinal disorders, and in many cases means nothing more than nature's attempts at heat dissipation. The importance of recognizing polypnea as a symptom of fever in infancy and childhood is great. If we do not do this, we may often be led, by the rapid breathing, away from the real cause of the disease. Fortunately for us as clinicians, there is a marked difference between the character of the polypneic breathing and the rapid respirations due to lung or heart disease. In polypnea the breathing is regular, easy and rapid, but is not, as it is in lung and heart disease, irregular, labored and accompanied by cyanosis.

[1] "Neurotic Disorders of Childhood," by B. K. Rachford, E. B. Treat & Co., N. Y., 1905.

# CHAPTER III

## EXAMINATION OF THE SICK CHILD

### HISTORY OF THE CASE

**Present Illness.**—The first step in the routine examination of the sick child is to listen attentively to the mother's and nurse's story of the child's present illness. In this short narrative the physician should obtain clues which will materially assist him in his own careful physical examination of the child. It is all very well for us to say that a full and complete examination should be made of every sick child, but this is manifestly impossible and in a sense unnecessary. It would be unwise and unnecessary, even if the physician had the time, to thoroughly examine in every case presented to him the blood, the sputum, the stools, the cerebrospinal fluid and the gastric contents, or to test the child's special senses and make a thorough examination of all of its reflexes. These and many other special examinations may be suggested by the story of the child's illness or by the subsequent careful physical examination to which the child is subjected. The diagnostic skill of the physician will largely depend on his ability to determine from the mother's story what should be the character of his preliminary examination and to judge from the findings of this examination, or the subsequent developments of the case, what special examinations may be necessary to clear the diagnosis in an individual case.

**Previous Medical History.**—Obtain the medical history of the child as to previous illnesses and especially as to similar attacks; the character of food it has been taking; the regularity of its habits in eating and sleeping; gastrointestinal disorders; acute infectious diseases; nervous disturbances and possible birth and subsequent injuries. Special inquiry along certain lines suggested by the story of the present illness may bring forth facts of great importance in the child's previous history which the mother would otherwise have overlooked.

**Family History.**—With the story of the child's previous and present illness in mind, the physician is better prepared to obtain from the mother such facts in the family history as may have a bearing upon the case. It is especially important to know the number of other living children and their general health; the number of the dead and the causes of their death. It may be necessary in an individual case to inquire carefully into the family history with reference to syphilis, tuberculosis, nervous disorders, gout, autotoxic attacks, gastrointestinal disturbances, a hemorrhagic diathesis, or other conditions in the ancestry of the child, which may throw light upon its present illness.

## PHYSICAL EXAMINATION

If the child is approached gently and tactfully it is possible to make this examination without resistance from the little patient. In order that the various steps of the physical examination may be made under the most favorable conditions it is wise to postpone the taking of temperature and the examination of the throat until the last, as these procedures may irritate the child.

The rectal temperature should be taken at the first examination and thereafter should be recorded at least twice a day until a diagnosis is reached. In many instances it is necessary to take the temperature at three-hour intervals, and, as a rule, it is advisable that a careful temperature record be kept until the child is convalescent. The value and significance of temperature observations have been outlined in the chapter on Fever.

The weight of the child should be ascertained, if possible, at the first examination. This observation helps the physician in determining the child's general physical condition and when compared with future weighings enables him to determine whether the child is losing or gaining weight. The importance of failure to gain in weight, as an indication of disease in the growing child, has been referred to in the chapter on Growth and Development.

**Inspection.**—More information may be obtained by inspection than by any other method of physical examination. That this may be thorough, the whole body of the child should be examined. The experienced eye can almost at a glance read the nutritional history of the child in the general picture which its nude form presents. It is important to note muscular development, general emaciation, the size and shape of the head, thorax and abdomen, skin eruptions, deformities, localized inflammations, and the presence or absence of the external signs of rickets, hereditary syphilis, gastrointestinal diseases, and anemia.

Face and Head.—The facial expression may tell of the presence of adenoids, paralysis, and cretinism and other forms of idiocy. The sunken eyes, pinched features and dull stare of the child indicate the extreme gravity of the illness. The small wasted face, large head and open fontanels tell the story of long illness from some chronic wasting disease. The rachitic head, the microcephalic head and the hydrocephalic head are readily recognized. Opisthotonos, stiffness of the spine and a position which shields the eyes from light suggest meningeal inflammation. If the head falls loosely in the direction gravity directs, and the spine shows by its abnormal flexibility a lack of muscular development, it may be inferred there is also a lack of mental development. Inspection of the throat and mouth commonly throws light on the character of the child's illness. One should observe the character of the tongue; local diseases of the tonsils, pharynx, and mucous membranes; the membrane of diphtheria; the scarlatinal sore throat, the enanthems of measles and of the other acute exanthe-

mata; abnormalities as to the formation of the teeth and the order of their eruption; hemorrhagic and other diseased conditions of the gums, and deformities in the shape and general contour of the palate. The skin should be carefully examined and one should note the presence and character of all rashes; desquamation of the skin and its character; cyanosis and its possible relation to dangerous cardiac and respiratory diseases; petechial and other hemorrhagic eruptions; jaundice as shown by the yellowish discoloration of the skin and conjunctiva; edemas, both general and localized, the former pointing to nephritis, the latter to urticaria and gastrointestinal disorders; marked pallor of the conjunctiva and skin, with or without edema, which may give a clue to grave blood lesions; syphilitic lesions about the anus; irritations, catarrhal inflammations and other abnormalities of the genitalia, and clubbing and blueness of the finger tips which may point to some grave lesion of the circulatory or respiratory apparatus.

The GENERAL POSITION of the child lying, sitting or standing gives much information. In pleurisy and pneumonia it may lie upon the affected side, holding its chest wall and evincing characteristic pain by its facial expression and by crying out when it is moved. In abdominal pain it usually lies upon its back with its thighs flexed upon its abdomen; this position is common in appendicitis, peritonitis, typhoid fever with abdominal distention, and in severe attacks of intestinal colic. In the standing position curvatures of the spine, bow-legs and other body deformities are readily detected.

RESPIRATORY MOVEMENTS should be carefully observed while the child is at rest. Marked dyspnea, with retraction of the chest, and dilatation of the alæ nasi may not only point to bronchopneumonia, but may indicate the seriousness of this disease, or the same symptom group slightly modified may be caused by an obstructive lesion in the larynx, such as may occur in diphtheria and catarrhal laryngitis. In very young infants difficult and interrupted breathing may result from nasal obstruction caused by syphilis, acute rhinitis, or retropharyngeal abscess. Slow and irregular respiratory movements may be due to intracranial disease.

**Palpation or Digital Examination.**—The HEAD and NECK are fruitful fields for this form of examination. One should note the shape of the head, size of the fontanels, thinness of the cranial bones, hypertrophied tonsils, enlargement of cervical lymphatics, and swelling of the parotid and other salivary glands. A digital exploration may reveal adenoid disease or other abnormalities in the pharynx.

Palpation of the CHEST may reveal the rickety rosary, or other bony deformities, the fremitus produced by the voice and by bronchial and sibilant râles, inequalities in the movements of the two sides of the chest, the location of the apex beat of the heart, and the cardiac thrill when present. By palpation one also determines the rate and character of the pulse and general and local enlargement of superficial lymphatic glands.

In the ABDOMEN one may discover by palpation localized or general

tonicity and resistance of the abdominal wall, so important in the diagnosis of appendicitis, peritonitis and other abdominal diseases; fluid in the abdominal cavity; enlargement of the spleen and liver; displacement and enlargement of the kidneys; tumors and other abnormalities. The liver normally extends one inch below the margin of the ribs when the child is lying upon its back; it is therefore readily palpable, and enlargements are easily detected. The spleen when easily palpable is enlarged and is a diagnostic sign of great importance in typhoid fever, leukemia, von Jaksch's disease, chronic forms of tuberculosis and malaria. Enlargement of the spleen may also occur in the acute infections, chronic gastrointestinal diseases and chronic forms of sepsis.

In the upper and lower EXTREMITIES one may discover tenderness and rigidity of the joints, deformities and lack of symmetry in development.

The method of examination for determining the rigidity of the SPINAL COLUMN is important. Curvatures of the spine are readily discovered when the child is undressed, but the character of these curvatures can only be made out by careful palpation. The organic curvature due to Pott's disease, which is permanent and rigid, is to be differentiated from curvatures due to false position and muscular weakness, such as are associated with rickets and other malnutritions. The nonflexibility of the spinal curvature of Pott's disease as compared with the flexible curvatures from other causes is one of the important diagnostic points. In making this examination the patient is placed upon the table face downward, the fingers of one hand are now gently inserted between the spinal processes over the curvature; with the other hand grasping and lifting the ankles of the child, its body is bent backward, and as the spine bends with the body the finger inserted between the spinal processes discovers no approximation of these processes, or, in other words, the spinal curvature remains rigid. In other forms of curvature the spinal processes are felt to close upon the fingers as that portion of the spine bends under the backward movement. Retraction of the head and opisthotonos, such as one sees in meningitis and certain other nervous disorders, may be discovered early by placing the hand under the head and lifting the body of the child while the neck remains rigid or bent backward. In marked cases the child may be lifted without bending the body by placing one hand under the head and the other under the legs.

**Reflexes.**—The PATELLAR REFLEX is one of the most easily developed and one of the most important of the deep reflexes. It may be elicited by placing the child in a sitting position with its leg flexed at the knee and hanging loosely from a chair, or in the infant the leg may be lifted by the hand as the infant lies in bed, so that the knee is sharply bent and the lower leg hangs loosely over the hand. With the child in one or the other of these positions the tendon just below the patella is struck sharply with the edge of the hand; in response to this the muscle contracts and the foot is thrown quickly upward. In certain diseases of the nervous system this reflex is absent, in others it is exaggerated. An exaggerated patellar reflex, especially if unilateral, is of great value in the late diagnosis of spastic

palsies due to cortical cerebral hemorrhage in early infancy, and the presence or absence of this reflex is of value in locating the site of a transverse myelitis. Its absence on both sides locates the myelitis at or below the second lumbar vertebræ. Its presence locates the myelitis above this point. The diagnosis of the myelitis itself must, of course, depend upon the other symptoms of this disease.

Babinsky's Reflex.—This reflex is present in some forms of meningitis and in all conditions which interfere with the conducting power of the pyramidal tracts. It is produced by drawing the tip of the finger across the plantar surface of the foot. It consists in a marked hyperextension of the great toe with a separation and perhaps extension of the other toes. This sign is of no value in children under two years of age, since at this

Fig. 4.—Palpating Spinal Curvature.

time of life, by reason of the lack of development of the pyramidal tracts, hyperextension of the great toe from plantar irritation is the normal response. Koplik has observed the Babinsky reflex more commonly in tuberculous than in other forms of meningitis, and Morse called special attention to the unreliability of this sign in young children.

Kernig's Sign.—The muscular resistance at the knee joint, which makes it impossible to extend the leg when the thigh is flexed at right angles to the body, is a sign of meningeal irritation. It is commonly found in all forms of meningitis and is sometimes present in meningeal irritation from acute toxic conditions such as pneumonia and typhoid fever.

**Percussion.**—As Hamill has emphasized, satisfactory results can be obtained by percussion only when the greatest care is taken to see that all the conditions are favorable. In order to distinguish the shades of sound elicited, quiet surroundings are absolutely necessary. To develop reliable percussion sounds the child should be in proper position. To percuss the

front of the chest the child should lie on its back on a firm bed or table; the two sides of its body should be symmetrically placed with the face directed upward and the spinal column straight. For percussing the back it should sit on the edge of a table or be held against the chest of the nurse with its face over her shoulder, great care being taken to have the spine and head straight and the body of the child as relaxed as possible. The distal phalanx of the middle finger of one hand is to be used as a pleximeter and placed firmly against the chest wall, with the middle finger of the other hand the distal phalanx thus placed is tapped quickly but gently. Great stress should be laid on very light percussion. The physician by experience will learn the force of the stroke that serves him to the best purpose. The value of the percussion note elicited at any point is judged largely by comparison with other percussion notes elicited in other portions of the chest. Hamill justly lays great stress upon these details. He also says the percussion of the chest of the normal infant or young child yields the following results: "On the right, from the clavicle to the fourth rib, one elicits the full normal pulmonary resonance. Below this point, owing to the decreased volume of lung, and the presence behind it of the liver, the sound becomes gradually dull and finally passes into the dull sound of the liver at the sixth rib. On the left side there is a relatively dull area behind and beneath the inner third of the clavicle, which sometimes extends outward to the mid-clavicular line, and always downward until it fades into the cardiac dullness." If percussion is skilfully done much information can be obtained concerning the pathological processes going on in the chest cavity. It is especially valuable in the diagnosis and differential diagnosis of diseases of the lungs, pleura and heart. By it also much information can be obtained concerning diseased processes going on in the abdominal cavity. Enlargement of the liver and spleen, and fluids, and tumors in the abdominal cavity may be mapped out by percussion.

FIG. 5.—PERCUSSION AND AUSCULTATION POSITION.

**Auscultation.**—The average practitioner obtains much more information by auscultation than he does by percussion. This is partly due to the fact that much less experience and skill is required to obtain information by auscultation. The position of the infant and child should be the same as that above described for percussion, as the auscultatory sounds may be readily changed by position. A binaural stethoscope with a small bell should be used, so that every portion of the chest wall including the axilla may be readily reached. In the auscultation of heart sounds the point of greatest intensity should be sought for. This is commonly at the apex or the base. The direction in which these cardiac murmurs are carried should then be carefully traced. Respiratory sounds on one side should be compared with the respiratory sounds at the same point over the opposite lung. In interpreting respiratory sounds it is important to remember that the expiratory murmur is more intense on the right side beneath the clavicle and over the spine of the scapula than it is on the left. The more intense respiratory murmur to the right of the sternum and beneath the clavicle is in contrast with the area of relative dullness in a somewhat similar position on the left side. It is important also to remember the rough inspiratory sound normally found over the lungs of infants and young children. This so-called puerile breathing, if slightly exaggerated, may be mistaken for bronchial breathing unless one remembers that the normally rough breathing of the infant is especially marked on inspiration, and the pathological bronchial breathing is more marked on expiration. If the physician knows the normal heart and lung sounds, auscultation, if carefully done, is of the very greatest value in the diagnosis of heart and lung diseases.

## SPECIAL EXAMINATIONS

**Examination of Urine and Stools.**—An examination of the URINE should be made, if possible, in every case; some obscure conditions may be cleared up in this way. It is impossible in an individual case to decide from other symptoms as to the necessity for examining the urine. This examination should include the presence or absence of bile, indican, indolacetic acid, albumin, the acetone bodies, pathological crystals, pus, pathological epithelium, blood and casts of various kinds.

INTESTINAL DISCHARGES should be inspected as a routine measure in all cases. This is especially important in children under two years of age. These discharges should be seen by the physician, as it is impossible for him to get accurate information concerning their character from the description of them given by the nurse. The following points should be observed: the reaction (if strongly alkaline protein putrefaction is indicated, if highly acid carbohydrate fermentation); the consistency—watery, spongy, pasty or formed; the color—green, yellow, brown, black or mottled; the presence or absence of curds (which if small and soft indicate undigested fat, if large and tough undigested casein)—other undigested food, mucus, blood and intestinal parasites. In some instances it may be necessary to make a

microscopical or chemical examination of the stools for the purpose of determining the presence or absence of blood, undigested food, the eggs of intestinal parasites, tubercle bacilli and pus.

Talbot says: "A microscopic examination of the stool gives very accurate information about the digestion of fat. Two stains are used, one on each of two coverglass preparations, alcoholic solution of Sudan III[1] and carbolfuchsin.[2] These stain the neutral fats, fatty acids and soaps differently. The following table shows these differences:

| Stain | Neutral Fat | Fatty Acids | Soaps |
|---|---|---|---|
| Sudan III | Drops staining red | Drops staining red or crystals which may or may not stain | Do not stain |
| Carbolfuchsin | Do not stain. Remain oily, colorless drops | Stain brilliant red | Stain dull red |

"After these two coverglasses are examined and the microscopic picture is clear, a drop of glacial acetic acid is allowed to run under the coverglass covering the Sudan III stain, is thoroughly mixed in, then heated until it begins to bubble. Care should be taken not to boil the preparation so much that all the melted fat will run off the slide. This process turns the soaps and neutral fats into fatty acids, which, while warm, appear as large red-stained drops, and, upon cooling, crystallize. This shows the amount of total fat in the stool, while the first two slides examined show the relative proportions of neutral fat, fatty acids and soaps. There is no way of differentiating neutral fat drops from fatty acid drops by Sudan III; it is, therefore, necessary to stain a second preparation with carbolfuchsin (see table) which does not stain neutral fat and does stain fatty acids. These tests are very simple, quick and valuable, giving accurate and often surprising evidence concerning the digestion of fat. They should always be used. An excess of fat can be easily determined and acted upon; absence of fat very often shows why the baby does not gain and always means that fat is not the cause of the indigestion. This rough method of estimating the relative proportion of neutral fats, fatty acids and soaps also gives an idea of the digestive functions. If there is an excess of fat most of which is split the digestion is normal and assimilation is abnormal; if the majority of the fat is unsplit or only partially digested both digestion and assimilation are abnormal."

**Tuberculin Skin Reactions.**—The reaction which results from inoculating the skin with Koch's old tuberculin has in recent years been very extensively used in the diagnosis of concealed forms of tuberculosis. These tests are of special value in childhood, since this is the period of life when latent or concealed tuberculosis of lymphatic and other tissues is so common.

[1] Sudan III powder, 95 per cent. ethyl alcohol. Saturated solution.

[2] Carbolfuchsin, such as is used in staining for tubercle bacilli. If the stain is too intense it may be diluted with equal parts of alcohol, 95 per cent.

The value and limitation of these tests are described in the chapter on Tuberculosis. The Moro inunction test is the simplest and the best for general use. In this test an ointment consisting of equal parts of anhydrous lanolin and "old" tuberculin is thoroughly rubbed into a portion of the skin about the size of a dollar; the abdomen is the site commonly selected. On the opposite side of the abdomen, in a similar location, pure lanolin is rubbed into the skin in the same vigorous manner; this is done as a control. A positive reaction is indicated by the appearance within twelve or twenty-four hours of an eruption over the area of skin into which the tuberculin was rubbed, while the skin on the opposite side into which the lanolin was rubbed remains normal. This eruption consists of small papules surrounded by a red areola, so that the whole area of the spot rubbed, and perhaps a portion of the surrounding skin, has an erythematous flush in which there is a maculopapular eruption. Von Pirquet's test is perhaps slightly more sensitive than the Moro test. It consists in scarifying the arm in three places, several inches apart; the scarification is made as in vaccination. Into the middle scarification Koch's "old" tuberculin is scratched or rubbed with the same technique used in Jennerian vaccination. A positive reaction is indicated by the appearance, within twelve or forty-eight hours, of an eruption of scattered papules within a dark, red zone, which occurs in and about the middle scarification, while the upper and lower scarifications show no reaction whatever. The inflammatory flush of the skin, which results from both the Moro and Von Pirquet tests, begins to subside within twenty-four or thirty-six hours after their appearance and thereafter quickly disappears. The severity of this skin reaction is not an indication of the extent or activity of the tuberculous process. A very pronounced reaction may be obtained in children who show no other signs of tuberculosis, and a negative reaction is commonly obtained in cases where the tuberculous process is active and associated with high fever and other signs of a destructive tuberculosis. The hypodermic injection of tuberculin and the dropping of a tuberculin solution in the eye will also give a prompt reaction in the latent and concealed forms of tuberculosis, but these tests have now largely fallen into disuse because the Moro and Von Pirquet tests are simpler, less disagreeable and are followed by absolutely no untoward results.

The chief objection to all of these tuberculin tests is that they are so sensitive that they give a reaction in all cases where there is the slightest focus of concealed tuberculosis. A negative reaction is of great value in excluding concealed forms of tuberculosis not associated with fever and other acute symptoms. A positive reaction is also of great diagnostic value in children, but the activity and extent of the tuberculous process must be determined by other signs and symptoms. It is evident, therefore, that, of these tuberculin reactions, the least sensitive will be of the greatest value from a clinical standpoint. The Moro test is slightly less sensitive than the others and for this reason it is of more practical value from a clinical standpoint.

PLATE I.

The Moro Tuberculin Skin Reaction.
(From Hamill, Carpenter and Cope).

**Blood Examinations.**—The ordinary blood examination should include an estimation of the amount of hemoglobin, the number and character of the red blood corpuscles, the color index, the number of white blood corpuscles and a differential count in which the relative percentages of the various kinds of white blood corpuscles are given. Such an examination is absolutely necessary to the diagnosis of diseases of the blood and of the blood-forming organs and is of great value in the diagnosis and prognosis of septic processes and of some of the acute infections.

**Lumbar Puncture (Quincke).**—A bacteriological examination of the cerebrospinal fluid obtained by lumbar puncture is of great value in the differential diagnosis of the various forms of meningitis. The operation is to be performed with clean instruments under aseptic conditions so that the

FIG. 6.—POSITION FOR LUMBAR PUNCTURE.

fluid when obtained will not be contaminated. In performing this operation general anesthesia, as a rule, is not necessary. The patient is placed on his side with his spine curved so as to bring prominently into view the spinous processes, and in this position he is to be firmly held by an assistant. The skin over the site of puncture is to be scrubbed with soap and water and washed with alcohol and a bichlorid of mercury solution. The operator then, with clean hands, inserts between the third and fourth lumbar vertebræ a clean trocar or cannula, about one millimeter in diameter, and by gentle pressure it is pushed directly inward for about three-fourths of an inch. If it meets with an obstruction it is to be withdrawn and reinserted. The entrance to the spinal canal is noted by the lack of resistance and by the flow of the fluid through the cannula. After the first few drops have escaped, from 20 to 40 c. c. of cerebrospinal fluid is allowed to flow

through the cannula and is caught in a sterile test tube. The needle is now withdrawn, the wound dressed with surgeon's adhesive plaster and the fluid subjected to a careful bacteriological examination, to determine if possible the microorganism causing the disease. The point of puncture is located by an imaginary line, passing directly backward over the spine between the iliac crests. With the child in position, the iliac crests on either side may be easily located, and the line passing between these two points directly backward over the spine crosses the third lumbar spine; the needle is inserted in the interspace below this spinal process. This operation is comparatively simple and is attended with little or no danger unless it be postponed until the child is *in extremis.*

**Röntgen-Rays.**—The pictures produced by Röntgen-rays are of the very greatest value in the accurate diagnosis of a large number of medical and surgical conditions, and the diagnostic field which has been opened up by this method is gradually becoming wider and wider. They are of special value in the diagnosis of the following conditions: injuries and diseases of bones and joints; kidney and bladder stones; foreign bodies in the intestinal canal, respiratory passages and other organs and tissues; diseases of the heart and lungs; enlargement of organs, such as the thymus gland, kidneys, liver, and deep-seated lymph nodes; collections of pus and other fluids in the pleural cavity, the pericardium, accessory sinuses of the face, the abdominal cavity and other parts of the body.

**Other Examinations.**—There are many other special examinations which may be necessary to clear the diagnosis in individual cases, such as the Widal reaction for the differential diagnosis of typhoid from other fevers; a bacteriological examination of the sputum for the purpose of determining the character of the infection in diseases of the respiratory passages; a bacteriological examination of the throat to distinguish diphtheria from other exudative inflammatory deposits; a bacteriological examination of inflammatory exudates in the pleura and other parts of the body, for determining the causative organism which has produced the infection.

## CHAPTER IV

### THERAPEUTICS OF INFANCY AND CHILDHOOD

Under this heading may be included all measures employed for the cure and prevention of disease. Diet, especially during infancy, is one of our most important therapeutic measures, but this is elsewhere discussed. It remains for us here to give an outline of other methods of treatment.

### MEDICINAL TREATMENT

**Drug Administration by Mouth.**—This is an important part of general therapeutics. By the administration of drugs a few diseases are cured;

many others are treated symptomatically so that symptoms are relieved, and the general course and character of the disease so modified that its duration is shortened and the chances for a favorable termination enhanced.

In almost all diseases the use of drugs is more or less indicated, either for their direct curative power, their favorable influence on the course and duration of the disease, or their control over disagreeable symptoms. Great as is the value of drugs skilfully and judiciously administered, the unnecessary and unskilful giving of medicines is almost of equal harm. Drugs should be given only when there is a decided probability that their administration will do the patient more good than harm. This rule of action is especially applicable to children under two years of age. In every sick infant, whatever may be the cause of its illness, the probability of gastrointestinal complications should be kept in mind. It is most important that the physician in giving drugs, during this period of life, should exercise the greatest precaution lest he produce some gastrointestinal disturbance and thereby add a serious complication to the existing malady. This precaution is especially important in the treatment of acute diseases of the respiratory passages and other acute infections. In these conditions I believe that infants are unwisely and unnecessarily medicated with opium, coal-tar products, nauseating syrups, such as ipecac and squills, and irritating ammonia preparations; all of which are of little or no value in the treatment, but, on the other hand, are capable of producing gastrointestinal and other complications which add a gravity to these diseases which they would not otherwise have had. In children under two years of age, and perhaps I may say in children of all ages, the physician before prescribing a drug should feel assured that there is a reasonable probability that the drug will exercise a favorable influence on the disease without producing harm in some other direction.

*Palatable medication* is one of the keynotes to success in pediatric practice. It is important that the most cordial and friendly relationship should exist between the physician and his little patient. Without this the physician falls short of having his visits and his ministrations accomplish the greatest possible amount of good to the child. The degree of friendship which exists between the physician and his small patients is largely a matter of his own making. If in his absence he instructs the mother to use her powerful influence over the child, to educate it into the belief that the physician's coming is an event to be looked forward to with pleasure, and if when the physician makes the visit he exercises the proper tact in his association with the child and uses his best judgment in the selection of proper remedies, which are either pleasant to the taste or their disagreeableness so disguised that the child will not associate anything unpleasant with the taking of medicines, then the relationship of the child and physician will be such that he can make his examinations and prescribe his treatment without engendering the child's ill-will.

The physician should remember that pleasant medication appeals to the mother almost as much as it does to the child. The forcing of disagreeable

medicines down the throat of a screaming and struggling child is an operation which neither the mother nor the child will submit to for any great length of time. A procedure of this kind in children who are acutely ill, and especially in nervous children, exercises a very unfavorable influence on the course of the disease, and the drug that produces more good than harm under these conditions must have a specific curative influence on the disease. While much stress is laid upon the necessity for pleasant medication and the tactful handling of the child so that a cordial relationship may exist, yet in following this line of action it is most important that the physician should not fail to make necessary examinations because they are unpleasant to the child; nor should he omit to give it some drug having a specific curative influence, such as quinin, mercury or antitoxin, simply because by its administration he may engender the ill-will of the child.

Opium is rarely to be administered to infants and children under two years of age. It may very occasionally be necessary to prescribe it during the second year of life for severe pain, such as occurs in earache. It should always be given with great caution and in small doses to children under five years of age.

Syrups of all kinds are contraindicated in infancy and are seldom necessary in children under five years of age. The only exception to this rule is perhaps in those cases where emetics are urgently indicated. As a vehicle for other medicines, glycerin, essence of pepsin and elixir of lactated pepsin are just as palatable as syrups, and are not as likely to disturb the stomach and cause intestinal fermentation. Quinin may be given in the form of euquinin or mixed with powdered chocolate to young children; older children may take it in the form of pills and capsules.

The following table gives the average dose and most common therapeutic indications of the drugs most frequently used in the treatment of diseases in children under three years of age:

| Drugs | Dose at 1 Year | Dose at 3 Years | Therapeutic Indications |
|---|---|---|---|
| Antipyrin | gr. ½–1 | gr. 1–2 | Nervous symptoms associated with fever. |
| Aspirin | gr. ½–1 | gr. 1–3 | Influenza, fever, nervous symptoms. |
| Belladonna tinct. | m ½–1 | m 1–2 | Coryza, pharyngitis, bladder irritation. |
| Bismuth subnitrate | gr. 2–5 | gr. 3–10 | Gastrointestinal irritation, diarrhea. |
| Calomel | gr. ½–1 | gr. 1–2 | Gastrointestinal disorders, febrile diseases. |
| Cascara sagrada ext. (aromatic) | m 5–10 | m 10–20 | Constipation. |
| Castor oil | dr. 1 | dr. 1–2 | As a preliminary cathartic in gastrointestinal disorders; acute febrile diseases. |
| Chalk comp. mixt. | dr. 1 | dr. 1–2 | Gastrointestinal irritability. |
| Chloral hydrate | gr. 1–2 | gr. 2–3 | Convulsive disorders. |
| Codliver oil | dr. ½ | dr. 1 | As a general tonic in nutritional disorders, such as rickets and tuberculosis. |
| Diastase (thick malt ext.) | dr. ½ | dr. 1 | As a digestive and tonic in nutritional disturbances, such as chronic intestinal indigestion. |
| Digitalis tinct. | m 1 | m 2 | As a heart tonic in cardiac and other diseases. |
| Guaiacol carbonate | gr. 1 | gr. 2–3 | Gastrointestinal disorders, tuberculosis. |
| Glonoin (nitroglycerin) | gr. 1/400 | gr. 1/300 1/200 | In condition of collapse and cardiac failure. |
| Hydrochloric acid dil. | m 1 | m 2–3 | Gastrointestinal disorders. |
| Iron sach. carb. | gr. 1 | gr. 2 | Anemic conditions. |

| Drugs | Dose at 1 Year | Dose at 3 Years | Therapeutic Indications |
|---|---|---|---|
| Ipecac syrup | dr. ½–1 | dr. 1–2 | As an emetic in spasmodic croup and other conditions. |
| Magnesia, milk of | dr. 1 | dr. 1–2 | As a laxative and stomach sedative. |
| Magnesium sulphate | gr. 10–20 | gr. 30 | As a cathartic in nephritis and other conditions. |
| Mercury with chalk | gr. ½ | gr. ½–1 | Syphilis and gastrointestinal disorders. |
| Mercury bichlorid | gr. 1/150 | gr. 1/100 1/50 | Syphilis. |
| Morphin sulphate | gr. 1/100 | gr. 1/40 | Severe convulsive disorders. (Given hypodermically) |
| Paregoric | m 5–10 | m 10–20 | Earache and other severe pain; rarely in intestinal disorders. |
| Pepsin essence | dr. ½ | dr. 1 | Digestive disorders, as vehicle for other medicines. |
| Phenacetin | gr. ½–1 | gr. 1–2 | Fever and associated nervous symptoms. |
| Potassium bromid | gr. 2–3 | gr. 3–5 | Nervous symptoms and as a cough sedative. |
| Potassium chlorate | gr. 1 | gr. 2–3 | Stomatitis. |
| Quinin sulphate | gr. ½ | gr. 1–2 | Malaria, influenza and as a general tonic. |
| Euquinin (tasteless quinin) | gr. 1 | gr. 3–5 | Malaria, influenza and as a general tonic. |
| Rochelle salts | gr. 10–20 | gr. 30–60 | As a cathartic in nephritis, enteritis and other conditions. |
| Salol | gr. 1–2 | gr. 2–4 | Gastrointestinal diseases, influenza and febrile conditions. |
| Santonin | gr. ¼ | gr. ½–1 | Intestinal worms. |
| Sodium benzoate | gr. 1–2 | gr. 3–5 | Influenza and other febrile conditions. |
| Sodium bicarbonate | gr. 2–3 | gr. 5–10 | Stomach disorders and autointoxications (acidosis). |
| Sodium bromid | gr. 2–3 | gr. 5 | Nervous symptoms, cough sedative. |
| Sodium iodid | gr. 1 | gr. 2–3 | Syphilis. |
| Sodium phosphate | gr. 5–10 | gr. 20–30 | As a laxative in gastrointestinal disorders, autointoxications and other conditions. |
| Sodium salicylate | gr. 1 | gr. 2 | Intestinal fermentation, rheumatism and tonsillitis. |
| Strophanthus tinct | m 1 | m 2 | Cardiac disease, pneumonia and conditions producing heart failure. |
| Strychnin sulphate | gr. 1/500 | gr. 1/200 | Pneumonia, myocarditis and conditions requiring a respiratory stimulant and general tonic. |
| Urotropin | gr. ½ | gr. 1–3 | Pyelocystitis and conditions requiring a urinary antiseptic. |

**Inunctions.**—The value of this method of administering drugs to infants and children is, I believe, not fully appreciated by the general practitioner. In the following outline I have quoted freely from a paper[1] on this subject which I published some years ago.

Inunctions are very much more efficacious in the treatment of disease in young children than they are in adults, for the following reasons:

1. In infants and young children the surface of the skin, in proportion to the body weight, is from four to six times greater than in adults. This brings the whole blood and lymph circulation in closer communication with the blood vessels and lymphatics of the skin, and makes it possible for drugs which are rubbed into the skin to pass quickly through the body and make their appearance in the urine, feces, bronchial mucus and other excretions.

2. In infants and young children the vasomotor mechanism is much more responsive to reflex stimuli than it is in adults, and for this reason the capillary circulation of the skin is made much more active by the application of heat and friction, as in the giving of inunctions. This facilitates

[1] *Amer. Jour. of Med. Sciencés*, Jan., 1909.

absorption and the ready introduction of medicines into the general circulation.

3. All lymphatic structures, including those of the skin, are relatively more active and functionally more important in the young child than they are in the adult. This facilitates the ready introduction of medicines through the skin into the lymphatic circulation.

4. In infants and young children nutritional problems are of vastly greater importance than they are in the adult, and for this reason it is of the utmost importance that the stomach and gastrointestinal canal should be kept in the best possible condition; consequently all drugs that can be advantageously administered in some other manner should be kept out of the stomach. This is especially true of drugs which are intended to influence general metabolism and to act upon diseased tissues remote from the gastrointestinal canal.

5. The disorders which can be treated most satisfactorily by inunctions, such as diseases of the lymphatic structures and respiratory passages, are much more common and much more severe in infants and young children than they are in adults. This fact very materially enhances the relative importance of the inunction treatment at this age.

6. Experiments demonstrate that certain medicines may be introduced into the circulating media of the body with great facility by inunctions, and that this result is more readily accomplished in infants and young children than it is in adults.

In the giving of inunctions the following technique should be observed. The skin of the chest and abdomen must be carefully washed with soap and warm water, and hot moist towels applied for a few minutes to warm and redden the skin. One drachm of the ointment should then be very carefully and gently rubbed in, for a period of five or ten minutes.

By this method I have demonstrated that guaiacol, iodin, oil of wintergreen and salicylic acid can be readily rubbed through the skin, appearing in the urine of the child from one and one-half to two hours after its application, thus showing that these drugs have passed through the blood and circulating media of the child, and have come in contact with its organs in every part of the body. The inunction method therefore is eminently fitted for the administration of these drugs in all diseases where they are indicated. Guaiacol, given in this way, may be used in the treatment of tuberculosis, influenza, bronchitis, bronchopneumonia and all diseases of the respiratory passages, and should to a large extent take the place of such expectorants as ammonia, squills, ipecac, and antimony. These latter drugs are not only of little or no value in the treatment of these diseases in infants, but they are capable of producing grave complications on the part of the gastrointestinal organs. Iodin administered by inunction is of positive value in the treatment of late syphilis, chronic glandular enlargements and subacute and chronic diseases of the respiratory passages. Oil of wintergreen and salicylic acid, given by inunction, are very valuable in the treatment of muscular rheumatism, acute and chronic articular rheu-

matism, chorea, tonsillitis, and endocarditis. The inunction method of administering mercury (blue ointment) has long been recognized as the safest and best method of administering this drug in the treatment of syphilis in young infants, and requires no elaboration here. Colloidal silver, within the past few years, has been administered hypodermically, by the stomach, and by inunction in the treatment of various forms of localized and general septicemias. The profession as a whole, I think, has come to recognize that this is a most valuable adjunct in our treatment of septicemia, and I, for one, after a large experience extending over a number of years, am firmly convinced of its efficacy. In acute enlargement of the lymphatic tissues of the neck, which may follow scarlatinal, diphtheritic and other forms of tonsillitis, I believe that this remedy, in the form of unguentum Credé, properly rubbed into the surrounding lymphatic tissues, is of very great value in preventing the spread of the disease and in controlling the localized sepsis. This drug can be given more efficaciously to infants and young children by inunction than in any other manner, and its value in combating general and localized sepsis is much greater in infants and children than it is in adults.

Guaiacol, iodin, oil of wintergreen, and salicylic acid, for inunction purposes, should be combined with anhydrous lanolin in the proportion of one drachm to the ounce, and the dosage of the ointment thus prescribed should be one small level teaspoonful thoroughly rubbed into the skin once or twice in twenty-four hours. Unguentum Credé should be given in the same dosage at least twice a day for a period of three or four days, and it is most important that it should be applied over a large surface of the body and should be thoroughly rubbed in.

## OTHER METHODS OF TREATMENT

The giving of medicines is a comparatively small part of the physician's duty. The questions of prophylaxis, diet, general hygiene, hydrotherapy, and special methods of treatment are more important than drug giving except in those comparatively few diseases for which we have specific medication. The giving of drugs should be considered as a valuable adjunct to other methods of treatment. This is especially true in the gastrointestinal, the respiratory and the acute infectious diseases, which make the vast majority of the illnesses of infancy and childhood. It is not my desire to belittle the importance of drug giving, but rather to emphasize the relative importance of other methods of treatment.

**Fresh Air.**—Fresh air is one of the most important curative agents we have for the treatment of disease. As Northrup has said, we mean by fresh air *outdoor air, cool, flowing air,* that is to say, the very freshest air which the child can obtain in the location in which it is being treated. The outdoor air which may be obtained on porches, and which comes into the sickroom through wide-open windows even in the downtown tenement districts of our large cities, is better than the indoor air, but it is not as good

as the outdoor air which can be obtained in the suburbs of our cities and in the surrounding country, and it is nothing like as good as the pure, open air of the mountains, seashore, and other locations far removed from the contaminating influences of cities. In the treatment of gastrointestinal and respiratory diseases the curative influence of pure, fresh, flowing air is of far greater value than drugs, and it is also of prime importance as a remedy in the treatment of almost all diseases of infancy and childhood. If the physician but realizes the importance of fresh, pure air as a thera-

FIG. 7.—FRESH-AIR WARD ESTABLISHED BY THE AUTHOR AT THE CINCINNATI HOSPITAL IN 1898.

peutic agent, his own common sense and judgment will direct him in arranging the details for the carrying out of this treatment. As Northrup has emphasized, this remedy should be given in large doses and throughout the whole of the twenty-four hours, but in doing this the physician must so instruct the mother and the nurse in such details as clothing, bedding and the location of the patient, either in the open or in rooms with wide-open windows, that while the child is getting the required amount of fresh air it may be kept warm in winter and cool in summer. In the chapter on Respiratory and Gastrointestinal Diseases further details as to this treatment are given.

**Hydrotherapy.**—Hydrotherapy in its various forms is one of the most valuable curative agents we have in the whole range of therapeutics. When water is applied to the surface of the body it reduces the temperature by abstracting heat and promoting evaporation; it stimulates the skin to increased activity; it acts kindly in controlling nervous symptoms, and, above

all, it has a general tonic effect, stimulating nutritional processes. In this it differs markedly from medical antipyretics.

Tub-baths.—Tub-baths have a wide range of applicability in the treatment of diseased conditions in infancy and childhood. Children, however, do not bear very cold tub-baths as well as adults, their young nervous systems are shocked by the sudden application of cold, and they do not readily react from the cold bath. It is advisable, therefore, to begin with a temperature of 100° F. and gradually add cold water until the temperature of the bath is reduced to from 80° to 90° F., according to the age of the child. In infants it is rarely necessary to reduce the temperature of the water below 90° F. In older children it may be reduced to 80° F. The patient should remain in the bath from five to ten minutes and then be rubbed dry and returned to bed. This remedy is especially indicated in the gastrointestinal diseases of infancy and is also of value in the treatment of typhoid fever, pneumonia, and other diseases in which the temperature runs high and is associated with nervous symptoms.

Sponge Baths.—These baths, when properly applied, reduce the temperature, quiet the nervous system, promote sleep, and have a tonic effect upon nutritional processes. They have much the same range of application from the therapeutic standpoint as the tub-bath. While somewhat less efficacious than the tub-bath, they have the advantage of being more easily administered and of producing less shock and excitement to the nervous system. In their application a rubber sheet should protect the bed, and on this the child, after having its clothing removed, is placed between two blankets. The entire body of the child is then sponged with water at 80° F. containing 5 or 10 per cent. of alcohol. During the sponging process, which may be continued for ten minutes, the parts of the body not being sponged are to be covered, so as to prevent unnecessary chilling of the body. The therapeutic effects of this measure are due not only to the application of cold water to the surface of the body, but to the rapid evaporation which is thereby promoted.

Cold Packs.—This is a measure used for the same therapeutic purposes as the tub and sponge baths. It has its widest range of applicability, however, in older children. It is a very effective measure for the reduction of high temperatures. The body of the child is surrounded with a sheet wrung out of water at about 90° F., and over this sheet, which clings closely to the child's body, ice is rubbed. This procedure may be continued for from ten to twenty minutes, depending upon the influence which the cold pack has upon the rectal temperature and upon the general condition of the child. During this process an icebag should be applied to the head. This bath may be modified by sprinkling the sheet with cold water from time to time, and fanning the body of the child so as to promote evaporation. Following these measures, the child is to be wrapped, sheet and all, in a blanket, a warm water bottle placed to its feet, while the icebag to the head is to be continued. After one-half hour the child is to be dried and returned to its bed.

The cold pack, cold sponging, or tub-bath may be repeated at intervals of from four to eight hours if necessary for the control of the temperature. It should, however, be remembered, as I have emphasized in the chapter on Fever, that children bear moderate and even high temperatures, as a rule, without serious inconvenience, and that unless the fever continues high and is associated with nervous and other symptoms it is not advisable to be too energetic in our efforts to reduce temperature. Where temperature reduction, however, is indicated as a therapeutic measure hydrotherapy in one of the forms above described is to be preferred to medical antipyretics.

Icecap.—Cold applied to the head in the form of an icecap is a simple and effective measure for reducing temperature, relieving headache and quieting general nervous symptoms. The icebag is of great value in the treatment of sunstroke, meningeal inflammation, acute inflammatory conditions of the heart and its membranes, appendicitis, acute parenchymatous tonsillitis, and in acute localized, congestive and inflammatory lesions in various parts of the body. In very young and delicate infants it should be cautiously applied, but apart from this it is a comparatively safe measure, productive of much good and rarely followed by untoward symptoms.

Cold Compresses.—Cold compresses, made by wringing a towel out of water of 75° or 80° F., and covering it with dry flannel, has the same therapeutic indications as the icebag. This measure is sometimes of considerable advantage in inflammatory diseases such as tonsillitis, pneumonia, pleurisy and endocarditis.

Hot Baths.—The hot bath is of great therapeutic value in the treatment of uremia, infantile convulsions, delirium and coma. It also has a very soothing and tonic effect in bronchopneumonia. It is one of our most effective measures for eliminating toxins in the acute infectious diseases and in the various forms of autointoxication. The child should remain in water of 110° F. for from five to twenty minutes, and should then be wrapped in a hot blanket for one-half hour. Following this it should be thoroughly dried and returned to bed.

Hot Compresses.—Hot compresses are of great therapeutic value in muscular rheumatism (lumbago), neuralgic headaches and other superficial neuralgias, abdominal pain and localized inflammations. They should be applied by wringing towels out of very hot water and placing these as hot as they can be borne to the affected part. The hot compress may then be covered with oiled silk and held in position with a dry towel.

Salt-baths.—Salt-baths are of some therapeutic value in the treatment of rickets and other malnutritions. A pound of salt should be added to six gallons of water at body temperature. The bath should last for ten or fifteen minutes and should be followed by vigorous rubbing or gentle massage; one such bath in twenty-four hours is sufficient.

Rectal Irrigations.—Rectal irrigations with cool water are of value in reducing the temperature of infants suffering from heat stroke and gastrointestinal disorders. A double rectal tube which permits of an in- and out-

flow and which can be passed well beyond the internal sphincter is to be used. In the beginning the temperature of the water should be about 90° F., and should be gradually reduced to 60° F. The irrigation may last over a period of ten minutes.

Water Taken by Mouth.—Water taken by the mouth reduces the temperature and is by far the simplest and best diuretic and diaphoretic. It is of the very greatest value during the first forty-eight hours in the treatment of all acute gastrointestinal diseases of infancy. It is indicated in all the acute infectious diseases, especially scarlet fever, and is of value in the treatment of all febrile conditions. It is of value in constipation,

Fig. 8.—Hypodermoclysis.

functional and nervous disorders, and all forms of autointoxication. During the first year of life the infant, as a rule, gets a sufficient quantity of water in its food, but, should it be necessary to cut down the food during this period of life, the deficiency should be made up by the addition of water. In infancy and childhood too little attention is given to the value of water in the conditions above named. It should be prescribed as any other remedial agent in all toxic and febrile diseases. Ice is a remedy of value in relieving irritability of the stomach, in allaying thirst, and in promoting the general comfort of the patient in febrile and gastrointestinal diseases, where for some reason water cannot be given in quantities sufficient to meet the demands of the patient. The ice in these cases should be held in the mouth until it melts. In young infants and children it may be

necessary to inclose the ice in a piece of gauze to prevent them from swallowing it.

**Hypodermoclysis.**—Hypodermoclysis, or the introduction into the subcutaneous tissue of a 0.6 per cent. common salt solution, is the most effective general stimulant and diuretic in all cases in which the body media is deficient in fluids. It is especially indicated in the profound prostration associated with acute gastrointestinal disorders (cholera infantum), severe cases of recurrent vomiting and severe hemorrhage. It may also be of value in uremia, cardiac failure, and in all cases where a powerful stimulant and diuretic is urgently indicated. One per cent. of bicarbonate of soda may be added to this salt solution in "recurrent vomiting" and other conditions where it is desirable to counteract an acidosis. The favorite sites for this injection are under the breast and the loose subcutaneous tissue of the back and abdomen. The salt solution should be sterile and should be introduced through a sterile needle under full aseptic precautions. In infants and children from six to ten ounces may be introduced at one time. If this is quickly absorbed the injection may be repeated, if necessary, within six or eight hours. The same strength of lukewarm salt or soda solution in cases of persistent vomiting may be of great value when introduced through a soft catheter into the colon, where it is readily absorbed. In this procedure the catheter may remain in position and the fluid be allowed to slowly escape by the drop-method or small injections may be repeated from time to time.

FIG. 9.—POSITION FOR NASAL DOUCHING.

**Nasal Douche.**—Washing out the throat, nose, and pharynx with a mild alkaline antiseptic solution is, as Caillé has emphasized, a measure of great prophylactic and curative value in diseases of these parts. The prophylactic value of this process in the prevention of all contagious diseases which affect the throat and respiratory passages, as well as of many other diseases, is not fully appreciated by the medical profession. In washing out the nasopharynx the child should sit upright, with its head inclined slightly over a basin. The mother or nurse, with an all-soft rubber nose

syringe, slowly injects the alkaline solution backward through the nose. By this procedure some of the solution comes out of the other nostril and a portion of it is carried downward through the pharynx into the mouth and is expectorated by the child into the basin. The direction of the tip of the syringe which enters the nostril should be almost directly backward. By this method mucus and mucopurulent secretions may be washed out of these parts, and their absorption thereby largely prevented. The danger of forcing fluid through the Eustachian tube into the ear and thereby causing an internal ear complication is slight as compared with the danger of infection from the purulent material if it is not dislodged and the inflammation modified or controlled by irrigation. Atomizers may be used for the same purpose, but they are much less effective than the nasal douche in the cleansing of the nasopharynx.

**Stomach Washing (Lavage).**—This therapeutic measure, introduced by Epstein, is of value in selected cases. Its value and range of application, however, are not so great in the child as they are in the adult. The older child is commonly so terrified by this measure that the resistance which it offers makes lavage of doubtful efficacy in all except the most urgent conditions, such as poisoning, or in conditions of unconsciousness, where even in the older child the tube may be introduced without resistance. In young infants, however, the ease with which this procedure is carried out gives it a much wider range of application. Its chief indication is for the relief of gastric irritability and for removing poisons from the stomach. It is urgently indicated in all cases of poisoning at any age and is of value for the relief of persistent vomiting associated with gastritis, chronic gastric indigestion, and pyloric spasm. This method, even in these cases, however, should not be abused. If the stomach is once thoroughly washed out and allowed to rest for four or five hours, water, with perhaps the addition of a little lime water, may be given for a number of hours until the gastric irritability has subsided, and then properly selected foods will be retained if the case be one of simple gastric irritability. If the vomiting persists after this careful procedure the case is probably one in which repeated stomach washings will be of little or no value.

The apparatus used consists of a funnel attached by means of soft rubber tubing to a No. 12 American catheter. A small piece of glass tubing is used to connect the rubber tubing with the catheter, so that the flow of fluids to and from the stomach may be observed. The child is to be wrapped in a sheet or blanket inclosing its arms and legs so that it may be firmly held by an attendant. It may be placed on a table, flat on its back, or may be held in a sitting posture, with its head upright against the body of the nurse. The finger of the left hand is now introduced into the mouth, depressing the tongue, and with the right hand the catheter is introduced into the esophagus and directed downward into the stomach. In the young infant there is no difficulty whatever in this procedure. The catheter on gentle pressure finds its way, without accident, into the stomach. The infant's mouth may be held open by holding the index finger between its

gums, and if it has both upper and lower incisors the catheter is to be pushed to one or the other side, so that the teeth will not impinge upon it. When six or seven inches of the catheter have been introduced, the funnel should be depressed for the purpose of siphoning out the contents of the stomach. A common salt solution (teaspoonful to the quart) or the same strength of bicarbonate of soda solution at a temperature of 100° F. may now be poured into the funnel, which is elevated to a sufficient height to allow the fluid to flow slowly into the child's stomach. When the stomach is full, as indicated by the contents of the funnel, both the funnel and tube should be depressed to a point which will allow the contents of the stomach to be siphoned off. This process is to be repeated a number of times, until the water which is siphoned from the stomach is clear, indicating that the stomach has been cleansed. If after introducing the tube into the stomach no fluid is returned, it is possible that the catheter has been obstructed by mucus or food. Under such conditions it is necessary to remove, cleanse, and reinsert the catheter. In withdrawing the catheter from the stomach it is important to make firm pressure on the soft tubing, so that the fluid contained in the catheter may not escape into the throat and larynx during the process of withdrawal. This is especially important in those cases where it is necessary to insert the catheter through the nose into the stomach rather than through the mouth.

Fig. 10.—Stomach-Washing.

**Gavage.**—Gavage, or the introduction of food into the stomach through a tube, should be preceded by preliminary stomach washing. After the stomach has been thoroughly emptied, certain foods, such as breast-milk, peptonized milk, albumin water, meat juice, and certain prepared meat preparations, may be introduced into and left in the stomach. Kerley has shown that food may sometimes be retained, when introduced in this way, in cases of persistent vomiting in infancy. Gavage may also be indicated

in the feeding of premature infants and in severe diseases, such as pneumonia, typhoid fever, and meningitis, where the condition of the patient makes it either impossible to administer food by the mouth, or where the irritability of the stomach is such that the food is not retained when given in this way.

**Rectal Enemata.**—The value of enteroclysis, or the introduction of normal salt solution into the colon to supply fluids to the tissues and organs in conditions of collapse, starvation, uncontrollable vomiting, profuse hemorrhage and nephritis has been referred to under the heading Hypodermoclysis. Ordinary enemata, however, are indicated for very different conditions. They have great curative value in acute and chronic enterocolitis and especially in those cases in which there is a marked tenesmus, with mucous and bloody discharges. The flushing of the colon in these cases with a normal salt solution washes away the fecal matter and mucus, exercises a local curative influence on the mucous membrane, diminishes the intestinal toxemia, and reduces the fever and nervous symptoms from which these patients suffer.

In the giving of rectal enemata for washing out the colon, a small rubber catheter should be inserted six or eight inches into the bowel; it is not necessary to introduce the tube higher. Through this catheter, which is attached to an ordinary fountain syringe, salt solution, one teaspoonful to a quart, is allowed to flow. The quantity of fluid introduced will depend upon the individual case, and varies, in the infant and young child, from a pint to a quart. When the catheter is removed pressure is to be made upon the buttocks and the child kept quietly in bed so that the fluid may be retained for a time, in order that it may more thoroughly dissolve the mucus and fecal matter and thereby more effectually cleanse the bowel. In giving the injection the child's position should be either on the back or left side, with the buttocks slightly higher than the body, and the fluid should be allowed to flow slowly into the bowel.

Rectal injections of salt water are also of great value for the relief of constipation, and when given for this purpose the smallest possible quantity of water which will produce the desired result should be used. This procedure produces less irritation than any of the local measures we have for the relief of constipation. It is especially valuable during the first year of life; in many cases it may be necessary to continue the use of small salt water injections over a period of many months, until the infant is old enough to have its constipation corrected by diet or other means.

Kerley has emphasized the great value of injections of small quantities of olive oil for the relief of constipation. This method is of special value in children over one year of age. From one to three ounces of olive oil should be injected into the colon before the child goes to bed, and should be allowed to remain there, if possible, over night, or until the oil excites sufficient peristalsis to produce an evacuation of the bowels. This is without doubt a remedy of great value in children over one year of age.

**Rectal Feeding.**—Rectal feeding is nothing like so successful or so fre-

quently indicated in the child as it is in the adult. Nutrient enemata, however, may be of value in uncontrollable vomiting, in acute gastritis produced by the swallowing of caustic chemicals, and in other conditions where it is impossible for a prolonged period to feed the child by the mouth. The food materials used for this purpose are soluble peptone preparations, egg albumin, peptonized milk, and dextrinized gruels. It may also very rarely be necessary to introduce into the colon certain stimulants, such as whiskey, brandy, digitalis, strophanthus, and strychnin. When this becomes necessary these stimulants should be well diluted with normal salt solution or dextrinized gruels. Rectal stimulation, however, in infancy and childhood is for the most part uncertain and unsatisfactory.

**Rectal Suppositories.**—Rectal suppositories are very largely used and their use very greatly abused in the treatment of constipation in infancy and childhood. Soap and glycerin suppositories, which are in such general use, are very effective for unloading the lower bowel, but their habitual use is productive of much harm. These suppositories in time produce more or less rectal irritation and predispose to hemorrhoids and fissures of the anus. The rectal irritation produced by these suppositories makes the sphincter more irritable and by causing its contraction aggravates the constipation. Suppositories are here mentioned, therefore, chiefly for the purpose of condemning their habitual use. An occasional glycerin suppository may be justifiable. Gluten suppositories are much less effective and are much less irritating to the rectum, and may be used at infrequent intervals with comparatively little danger of producing disease of the rectum.

Suppositories are very commonly used in the adult as a vehicle for giving opium, belladonna, and other remedies, but they are very rarely used for this purpose in the child. Collargum, however, may be very advantageously given in this way to older children in the treatment of general septic conditions.

**Rest and Muscular Exercise.**—Rest in bed and muscular exercise are therapeutic measures of the greatest importance in the cure of disease. In order, however, that the best results may be obtained from these measures they must be prescribed with a precision that requires more skill and acumen on the part of the physician than are required for the giving of drugs, or for the proper use of any other therapeutic measure. It should further be remembered that to obtain the best results the fresh air treatment must be combined with these agencies in the treatment of disease.

Rest Cure.—Under this heading may be included rest in bed, which is absolutely necessary in the treatment of pneumonia, scarlet fever, typhoid fever, and all the acute infectious diseases. In appendicitis and acute inflammatory diseases involving the peritoneum, the rest in bed must be prolonged until convalescence is thoroughly established. In acute nephritis the patient must be confined to bed until the urine findings are normal. In tuberculosis, associated with fever, the patient should rest in a reclining position, or in a comfortable chair in the open air, until the active tuber-

culous process is under control. In acute diseases of the heart, such as myocarditis, endocarditis, and pericarditis, prolonged rest in bed is the most valuable therapeutic agent we have. The temporary recovery as well as the future welfare of these cardiac cases depends to a large degree upon the skill with which the physician prescribes the period of rest and the gradual return to light exercise. In many of the functional nervous disorders of childhood rest in bed under quiet surroundings and under the careful direction of a tactful nurse is of great value. Northrup has emphasized the value of this method of treatment in neurotic infants suffering from indigestion, sleeplessness, and other nervous symptoms.

Massage.—Massage is a valuable therapeutic agent. It gives tone and strength to muscles which have weakened under injury or disease; it improves the circulation in superficial muscles and other tissues; it stimulates the functional activity of certain internal organs, such as the liver and intestines; it acts as a general tonic, stimulating normal processes of metabolism; it has a sedative effect on the nervous system; and it promotes the elimination of toxins, especially autotoxins.

General massage is of special value to children when more active forms of exercise are contraindicated or when they are suffering from deformities which cannot be reached by active exercise. It may be indicated in recurrent vomiting (interval treatment), general malnutritions, chronic tuberculosis, chronic anemia, and in all conditions in which the muscles of the child are poorly developed. It is indispensable for the proper treatment of spastic and flaccid paralyses associated with cerebral and spinal palsies. In these conditions massage is to be associated with proper orthopedic treatment, not only for developing the muscles, but for correcting the deformities. In chronic constipation massage is of special value. In this condition deep massage beginning at the cecum is to follow the line of the colon to the sigmoid flexure; the so-called cannon-ball massage, which consists in rolling a covered iron ball weighing three or four pounds around the circumference of the abdomen in the direction of the colon, is a valuable expedient in some cases.

Passive and Resisted Movements.—Passive and resisted movements are of value in the correction of deformities and the development of muscles weakened or paralyzed by injury or disease of the nervous system. The contractures which occur in cerebral palsies should receive this form of treatment.

Gymnastic Exercises.—Gymnastic exercises, when skilfully directed and carefully carried out, are of very great value in correcting spinal curvatures and other deformities not due to organic disease of the bones or nervous system. Under a skilful physical director these exercises may give symmetry of development to the body.

Breathing Exercises.—Breathing exercises, when properly done in the open air, increase the respiratory capacity, carry more oxygen into the lungs and blood, promote the elimination of carbonic acid, and thereby act as a general blood tonic, improving nutritional processes.

It is of importance that every child should be taught how to breathe so as to develop his full lung capacity. This can only be done by combining with deep inspiration and slow expiration certain body and chest movements, which will bring into play not only the diaphragm and intercostal muscles, but all the accessory muscles of inspiration. If the habit of proper breathing is acquired during childhood it commonly becomes a habit which lasts through life.

Breathing exercises are of value in undersized, malnourished children with poor lung capacity. They are especially indicated in chronic anemia and chronic tuberculosis, as well as in children who, by reason of enlarged tonsils and adenoids, have poorly developed chests.

Outdoor Play.—The ordinary outdoor games, such as ball, tennis, skating, running, jumping, etc., are vastly superior to the forms of exercise above noted for the development of the normal child. Outdoor games are also of value in promoting the physical and incidentally the mental development of children who from heredity, environment, or chronic illness are below the normal in physical development. In many instances, however, outdoor sports when directed only by the child's instincts and desires are too strenuous or otherwise unsuited to bring about the best results in development in an individual case. In such instances the physician may prescribe modified outdoor sports under careful supervision, and that may be combined with gymnastic exercises, breathing exercises, passive and resistant exercises or massage, according to the demands of the individual case. Exercises, manipulations, and movements to accomplish definite and specific results must be carried out under the direction of an instructor, and massage, passive and resisted movements must be intelligently done by one who understands the results which the physician seeks to obtain in the individual case.

**Psychotherapy.**—In the treatment of neurotic disorders in children, as well as in the adult, psychotherapy is a valuable therapeutic agent. The young child suffering physical pain, or with wounded feelings, crying bitterly, rushes into the arms of his mother, she presses him to her breast, kisses away his tears, and by a kind and tactful word makes him forget his woes, directs his plastic mind into other channels of thought and sends him away laughing and happy. This is an everyday example of the powerful influence which suggestion may exercise over the emotional, imaginative, and imitative mind of the young child. By word pictures alone the imaginative mind of the child may be made to see terrifying objects which may cause a sleepless night. On the other hand, the sleepless, nervous child, whose mind has been excited by an overwrought imagination or by undue nervous strain before going to bed, may be quieted and ofttimes put to sleep by reassuring words from a forceful, tactful, and sympathetic mother, who knows how to use the control which the unlimited confidence of her child has given her over its emotional nature.

Nervous habits, such as stammering, habit-spasm, masturbation, nail-biting and dirt-eating, may be contracted by association with other children

having these habits. The imitative nature of the child makes it possible to influence its immature nervous system for good or evil by favorable or unfavorable surroundings. In the treatment of the above-named nervous habits it is important, therefore, that the patient should not be associated with nervous children or be under the care of a nervous, emotional nurse. It may be necessary to tactfully disregard or forcibly control certain nervous habits, or, again, it may be wise to ingenuously disclaim the existence of nervous symptoms. In this manner the child may be surrounded by an atmosphere which will cause it to be interested in outside things and less intent upon its own nervous condition. At times it may be necessary to separate a nervous child from its surroundings and place it under the exclusive care of a kind, firm, tactful nurse. The child very quickly realizes that it cannot enlist the sympathy of the nurse, as it did its mother's, by emotional outbursts. Under such conditions the kindly indifference, the tactful firmness, and the gentle attentiveness of the nurse may exercise a most helpful influence in the cure of hysterical and kindred neurotic disorders of childhood. It is difficult to formulate rules to guide the physician in the use of psychotherapy as a therapeutic measure. It is important to remember that children are emotional, imaginative and imitative, and that their immature minds are very receptive and easily influenced by suggestion from those in whom they have confidence. The physician who has a good working knowledge of child nature, the skill to apply this knowledge in controlling the child's emotions, and the tact to accomplish this through the co-operation of the mother will find many opportunities for using psychotherapy as a therapeutic measure, not only in the ordinary neuroses of childhood, but in many other diseases in which nervous symptoms are prominent.

**Vaccine Therapy.**—In 1903 A. E. Wright demonstrated the presence of certain substances in blood serum, which by their action so disabled bacteria that phagocytic cells could more easily take them up and destroy them; these substances he called opsonins. Previous to this discovery it was known that certain other bodies in the blood had the power of destroying or limiting the action of bacteria: "the agglutinins" conglomerated bacteria; "the bacteriolysins" and "bacteriocidal substances" destroyed them. All of these, including opsonins, are called antibodies, because of their antagonism to bacteria and other foreign cells that happen to find an entrance into the body media.

Wright believes that nature by the development of a specific opsonin for each bacterium makes it possible for the phagocytes to limit the course and modify the severity of both local and general bacterial infections. He also believes that both natural and acquired immunity against bacterial infection depends much more upon the opsonic content of the blood than upon any of the other antibodies. These views naturally led Wright to the development of a method which had for its purpose the cure of bacterial diseases by artificially increasing in the blood serum of the patient the particular opsonin which assisted in the phagocytosis of the specific microorganism causing the infection.

Opsonic Index.—The opsonic power, or opsonic content of an individual patient's blood serum, when divided by the opsonic power or opsonic content of the blood serum of a normal individual, will give the opsonic index of the patient's blood serum. The opsonic index thus obtained may in skilful hands, as Wright has demonstrated, be of value in an individual case in determining the necessity for vaccine treatment, as well as the dose of the vaccine to be administered, and subsequently the influence which the dose of vaccine has had in increasing the opsonins in the blood.

Accumulated experience with the vaccine treatment has demonstrated that successful vaccine therapy may be carried on without the use of the opsonic index, and this has brought vaccine therapy within the scope of the general practitioner.

Without Opsonic Index.—In giving bacterial vaccines with no knowledge of the individual patient's opsonic index it is advisable to begin with a small dose. The size of the subsequent doses as well as the length of the interval between the treatments may then be determined by the clinical reaction. The "clinical reaction" is manifested by a slight rise in temperature, a feeling of malaise, and by a slight exaggeration of existing symptoms. After twenty-four hours this is followed by a fall in temperature and general improvement in all the symptoms.

As a guide to the giving of bacterial vaccines without the use of the opsonic index, the following rules may be observed:

1. Begin with a small dose.

2. If no "clinical reaction" whatever occurs, and no improvement in the patient's condition takes place within three days, a second and larger dose may be given. This rule of action may be followed until a "clinical reaction" does occur, or until the patient commences to improve under the treatment. If the patient is improving under a certain dose of vaccine, even though no "clinical reaction" occur, the same dose should be continued at a five to seven-day interval. Wright does not believe it advisable to elicit the "clinical reaction" as an indication that the size of the dose is sufficient. Improvement in the symptom group is a more reliable indication.

3. If the "clinical reaction" follows the giving of a dose of vaccine and this is followed by improvement in the patient's symptoms, a second and smaller dose should be repeated in six or seven days, and this rule of action is to be continued as long as vaccine therapy is indicated.

4. If a "clinical reaction" is not followed by improvement in the patient's condition, vaccine therapy is not indicated and may do harm in that particular case.

Bacterial Vaccines.—Bacterial vaccines consist of sterile mixtures of dead bacteria in salt solution. Each c. c. of these vaccines contains a definite number of dead bacteria. The dose of the vaccine is regulated by the number of dead bacteria one desires to give in an individual case. There is as yet no way by which the potency of the dose may be accurately standardized. One does inject a definite number of killed bacteria, but the

potency of this dose depends not alone upon the number of bacteria injected, but also upon the virulency of the particular bacterial culture from which this vaccine was made. This inaccuracy in dosage, together with our lack of knowledge as to the manner in which an individual patient will react to different vaccines, makes the proper initial dose in every instance more or less problematical; for these reasons it is desirable to begin with small doses, which experience has taught are safe.

The best results from vaccine treatment are obtained by the use of autogenous vaccines, that is to say, vaccines which are prepared from a culture of the individual organism causing the disease. The difficulty in the technique, however, makes it rarely possible for the general practitioner to use autogenous vaccines; this is possible only when he has at his disposal a well-equipped laboratory and the assistance of a competent bacteriologist. This difficulty has brought into more or less general use the stock vaccines now on the market, which can in many instances be relied upon to produce satisfactory therapeutic results if the practitioner has the facilities at hand for making an accurate bacteriological diagnosis. For example, if it is definitely determined that a given infection is due to a certain staphylococcus, or that there is a double infection in which both the staphylococcus and gonococcus play a part, then the physician by the use of the respective stock vaccines for combating infections caused by these organisms may hope to obtain satisfactory results.

The safe initial dose of the staphylococcus vaccine, during the first year of life, is about 5,000,000 dead staphylococci; during the second year of life about 10,000,000; during the third year about 20,000,000; during the fourth year about 30,000,000; increasing 5,000,000 each year thereafter up to the tenth year of life, when it is about 60,000,000. The safe initial dose of the gonococcus vaccine is about one-tenth that of the staphylococcus vaccine above given, and that of streptococcus and pneumococcus vaccines about one-fifth.

*Therapeutic Indications.*—Vaccines are indicated in localized rather than in general infections, and in subacute and chronic rather than in acute infections. It would appear from the present literature on this subject that vaccines are of value in acute general infections only when the infection is mild or when the vaccine is given *very early* in the disease. In a severe, acute, general infection there is danger that the vaccine may aggravate the disease and not be followed by a favorable reaction. In these cases the tissues under the stimulation of the general bacterial invasion have already furnished the "high tide" of opsonins for the individual case and cannot further be stimulated to produce an increased quantity of these curative agents. The definite field of vaccine therapy has not as yet been determined, but there is no doubt that it exercises a curative influence in a group of cases which do not yield readily to other methods of treatment.

Localized staphylococcic infections, especially those due to the staphylococcus pyogenes aureus, such as occur in *furunculosis*, pustular acne, sycosis, cystitis, carbuncle, and osteomyelitis, may be favorably influenced,

and recovery accelerated by the use of the *staphylococcus vaccines*. While the above named conditions are very commonly due to staphylococcic infection, it is advisable to determine this fact by bacteriologic examination before using the vaccine. Especially good results have been obtained by this method in the treatment of chronic furunculosis. In the treatment of pustular acne with infiltration a combination vaccine containing both dead staphylococci and dead acne bacilli has been used with advantage.

*Koch's tuberculin* has been successfully used as a vaccine in the treatment of tuberculosis, but it is of little value in the treatment of this disease during the first year of life. Localized non-febrile forms of chronic tuberculosis (not meningeal), in children over two or three years of age, may be greatly benefited by this treatment, provided it is carried out as recommended by Trudeau. One should begin with very minute doses, .001 mg., and increase very gradually. The dose should be graduated so as to produce a very slight clinical reaction. A second dose should not be given for some days after all effects of the previous reaction have passed away. This treatment should be continued only in those cases that continue to improve. It should not be hastened, as it requires many months to get results. The tuberculin treatment may hasten the cure of cases which by reason of outdoor life and proper food have begun to improve, and have thereby indicated that the opsonic content of their blood may be increased by vaccine treatment.

*Gonococcus vaccine* is of little value in the treatment of acute gonorrhea except perhaps in the gonococcic vulvovaginitis of young infants, but in chronic cases, both in children and adults, the published reports indicate that it may shorten the course of this disease. The most important field for the gonococcic vaccine is found in the complications which are due to the action of this organism in other parts of the body. In subacute and chronic gonorrheal arthritis, and in chronic suppurative processes produced by gonococci in any part of the body, the proper surgical treatment of these conditons will much more readily bring about a cure when combined with the use of the gonococcus vaccine.

*Streptococcus vaccine* is, as a rule, not indicated in acute general streptococcic infections, but in all localized streptococcic infections, especially if they be subacute or chronic, this vaccine may be of value in facilitating a cure. Such localized streptococcic infections occur most commonly in association with, or as sequels of, influenza, epidemic grippe, tonsillitis, scarlet fever, tuberculosis and other acute infectious diseases. The ordinary sites of localized streptococcic infection are lymphatic glands, the serous cavities, the accessory sinuses of the nose and the subcutaneous tissues. In properly selected cases of this character the streptococcus vaccine may be of value. In the treatment of streptococcic infections autogenous vaccines are much to be preferred, because of the great variety of strains of this organism.

*Pneumococcus vaccine* is of little or no value in the treatment of general pneumococcic infections or of pneumococcic pneumonia during the

acute stage of the disease. It may be of value, however, in chronic forms of pneumococcic pneumonia in which there is delayed resolution; in subacute or chronic pneumococcic empyema, and in all localized pneumococcic infections, such as may occur in the joints, the internal ear, accessory sinuses of the nose and the urinary bladder.

*Typhoid vaccine* has been successfully used as a prophylactic measure against typhoid fever; its value, however, in the treatment of the disease itself has not been demonstrated. The vaccine treatment of erysipelas, scarlet fever and other acute infectious diseases has not as yet been followed by sufficient success to demonstrate its value during the acute stages of these diseases.

*Coli vaccine* is a valuable remedy in infection of the urinary tract due to this organism. It may also be given with some hope of success in colicystitis and catarrhal jaundice.

Antiserums.—It has been a well-known fact for many years that most of the acute infectious diseases are self-limited, run a more or less definite course, and are cured by the development in the body of the animal of antidotal substances acting on or destroying either the bacteria or their poisonous products, and this process by which nature effects a cure of these diseases is followed by a period of permanent or temporary immunity from the specific bacterial disease from which the animal was suffering. It seemed more than probable that if antiserums containing these antidotal substances could be artificially manufactured by immunizing horses or other animals by the injection into their blood or other tissues of certain bacteria and their poisonous products, these antiserums would act specifically in assisting nature in terminating or curing the specific infection caused by the microorganism which was used to produce the antiserum. A vast amount of experimental work in recent years has been directed along these lines with the result that certain antiserums have been produced which act specifically in the destruction of the parasite and its poisons without producing any injurious action on the body cells. The discovery of these antiserums furnishes the most notable therapeutic advance in the history of medicine.

The development of antiserums for the cure of bacterial diseases is but yet in its infancy and many of the problems connected with their manufacture are yet to be solved. Nevertheless, the achievements in this field of experimental medicine are nothing short of marvelous, and the future promises that achievements in this field of therapeutic research will be even more brilliant than the results which have been already obtained. At the present time it appears that there are at least two distinct classes of antiserums.

The first is represented by the diphtheria and tetanus antitoxins. In these diseases the bacteria acting within a localized area of infection excrete soluble poisons which are distributed through the circulating media of the animal to all parts of the body, producing a dangerous and frequently a fatal toxæmia. The antiserums which are produced for the

cure of these diseases are essentially *antitoxins* which combine with and neutralize or destroy the poisonous effect of toxins, thus giving nature an opportunity to furnish such antibodies as are necessary to effectually terminate the disease. The brilliant results which have followed the treatment of diphtheria by antitoxin are fully discussed in the chapter on Diphtheria.

The second class of antiserums is represented by the antimeningitis serum, the use of which has been followed by such brilliant results in the treatment of meningococcus meningitis. This serum is an *antiendotoxic* serum and is bacteriolytic rather than antitoxic. It acts primarily by destroying the meningococcus itself and secondarily by neutralizing the action of the toxins set free by the destruction of this microorganism. The toxins of the meningococcus, unlike those of the diphtheria bacillus, remain united with the microorganisms which have produced them, and are not therefore thrown into the body media, producing a general toxemia. The antimeningitis serum depends for its bacteriolytic action upon the opsonins which it contains. These opsonins prepare the meningococcus for ingestion and destruction by the leukocytes. The brilliant results which have been obtained by this serum are given in detail under the treatment of Meningococcus Meningitis.

Twenty years ago I wrote as follows:[1] "In imitating nature experimental clinical medicine has a promising field here opened for original work. In the cure and prevention of disease the experimenter may use one of two substances.

1. The chemical substances produced by the body cells.
2. The chemical substances produced by bacteria.

Let us first note what results we may expect from the use of chemical substances produced by the body cells in curing disease.

(*a*) By injecting these substances in sufficient quantity into the body of a healthy animal we would expect to confer complete temporary immunity to the particular bacterium that induced their formation. But this immunity would gradually disappear with the excretion of the cause on which it depended.

(*b*) By injecting these substances in sufficient quantities into the body of an animal sick of the disease we would expect it to act as a true specific in the cure of the disease.

If, therefore, we could obtain the various chemical substances with which nature cures the self-limited diseases and with which she confers a temporary immunity against them we would have the means not only of curing but of preventing these diseases.

Let us now note what beneficial results may be expected from the introduction of the products of bacteria. Whatever action these may have must depend upon their power of exciting the body cells to the production of substances that either destroy bacteria or neutralize their products and

[1] "Mechanism of Immunity," *Philadelphia Medical News*, April 23, 1892.

thus terminate the disease and confer immunity. The disease is here terminated and immunity conferred not by the products of the bacteria, but by the products of the cells in the same manner as when the cellular instead of the bacterial products are introduced. "The only difference is that in the first instance the cellular products are formed in the body of one animal and introduced as a curative or prophylactic agent into the body of another animal and in the second instance the cellular products are formed in the body of the animal for the purpose of conferring immunity, * * * or terminating a self-limited disease."

In the paper from which the above quotation is made I clearly outlined the possibilities and probabilities of the vaccine and antiserum treatment of bacterial diseases.

# SECTION II

## *THE NEW-BORN*

## CHAPTER V

### THE CARE OF PREMATURE INFANTS

**Physical Peculiarities of the Prematurely Born.**—Infants born prematurely differ from full-term infants in the comparative lack of functional development of many of their most important organs. By reason of this lack of development they are to a greater or less degree, depending upon the stage of prematurity, unfitted to live under the ordinary conditions of home and hospital life. The most important and the most serious defect of the premature infant is the lack of development of its *nervous system,* and especially the undeveloped state of its heat-regulating apparatus. Its thermogenic centers are so poorly developed that it is unable to produce the requisite amount of heat to maintain a normal body temperature. The body temperature of the unborn infant is that of its mother, but this heat has been largely furnished by the surroundings of the infant and is not due to the activity of its own heat-producing centers. At birth it is ushered into a room temperature between 70° and 80° F., and its body temperature rapidly falls and may within a few hours be as low as 85° F. The deficiency, however, of its heat-producing centers, which makes it dependent upon external heat for the maintenance of a normal body temperature, is not the only defect in its heat-regulating mechanism. The prime defect in the nervous system of the premature infant is the almost complete lack of development of its inhibitory functions, and this lack of inhibition is especially important in its influence, or rather lack of influence, on the heat-dissipating mechanism. This part of the heat-regulating apparatus is under so little control from higher nerve centers that the body heat of these infants is very rapidly dissipated when they are transferred from an intrauterine temperature of 99° F. to a room temperature of 70° F. The thermoinhibitory centers of these infants also exert but little or no control over the thermogenic centers and as a result of this maladjustment of the heat-regulating mechanism it is very difficult to maintain a normal body temperature in these infants. Artificial heat when applied with the purpose of supplying the deficiency in body heat may produce

dangerously high temperatures. I have seen the rectal temperature of a premature infant raised to 109° F. by a careless application of artificial heat, and in the same infant when the artificial heat was removed I have seen the temperature drop, within a few hours, to 93° F. The lack of development and instability of the heat-regulating mechanism of the premature infant predispose these infants to dangerously high and low temperatures from insignificant causes. Artificial heat, which is necessary to main-

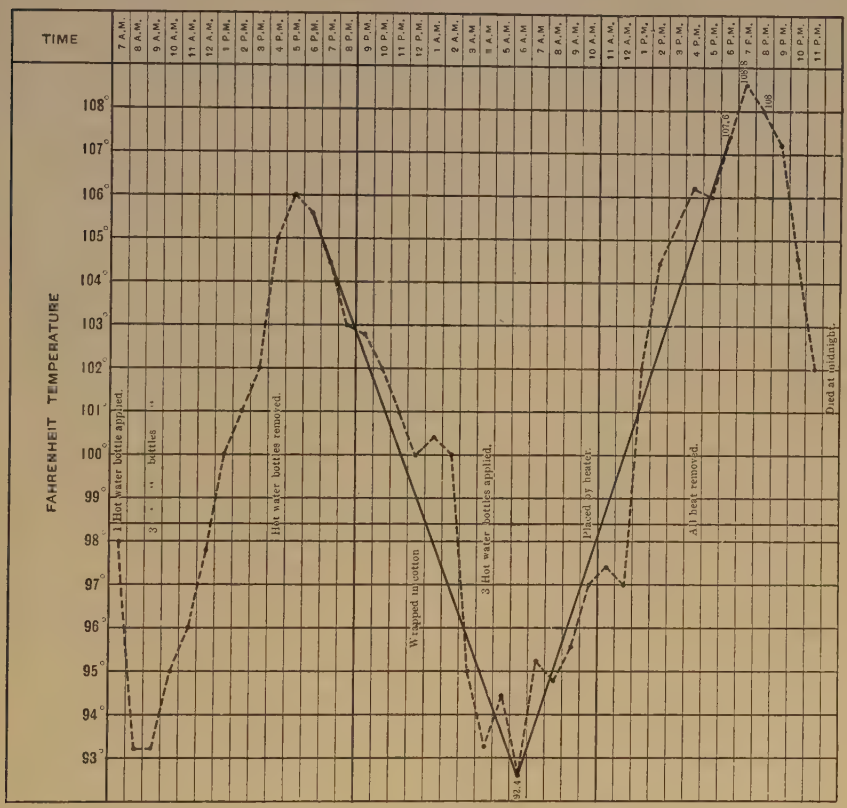



FIG. 11.—TEMPERATURE CURVE SHOWING INFLUENCE OF ARTIFICIAL HEAT ON PREMATURE INFANT.

tain a normal body temperature in these infants, should be applied under the most careful supervision.

The respiratory centers are also very imperfectly developed. Many of these infants are more or less asphyxiated at birth and in all of them the respiratory movements are feeble and shallow. The respiratory centers in these cases do not respond so energetically and satisfactorily to external reflex stimuli as they do in the full term infant.

The higher nerve centers are also imperfectly developed, so that these infants are somnolent, quiet and motionless a great portion of the time;

when aroused from their stupid condition they whine faintly, instead of uttering the lusty cry of the normal infant.

The reflex centers in the spinal cord, as well as those of the brain and medulla, are comparatively undeveloped. As a result of this there are comparative lack of muscular movement in the arms and legs, deficient tone in the muscles of the extremities, constipation or insufficient evacuation of the bowels.

The second most important defect in development is to be found in the *digestive tract.* The digestive organs of premature infants are, as compared with those of the normal child, to a greater or less degree physiologically incompetent. The degree of this physiological incompetency will depend upon the stage of prematurity. Infants born between the sixth and seventh month may manifest little inclination to suck; this reflex function is but feebly developed, and swallowing may be accomplished with difficulty. In infants born nearer full term there is usually no disturbance of the reflex acts of sucking and swallowing, but the digestive ferments are diminished in quantity and the digestive capacity is therefore markedly diminished. The degree of functional development of the gastrointestinal organs of the premature infant is of the very greatest importance from the standpoint of prognosis, since its life as well as its development depends upon its capacity to digest and assimilate sufficient food, not only to furnish body heat, but to supply nutrition for its growth and development. Among the prominent and discouraging symptoms, therefore, are those which arise from gastrointestinal indigestion. The meconium is passed for five or six days, but after this normal milk stools should begin to appear. If, however, the infant fails in its digestive capacity and the discharges indicate a gastroenteric indigestion, the prognosis becomes very grave indeed.

The *susceptibility to infection* is increased with the degree of immaturity of these infants and this susceptibility depends not only upon the lack of resistance to pathogenic microorganisms due to a lack of development of the defensive mechanisms by which normal infants offer more or less resistance to invading bacteria, but also to the ease with which these microorganisms find an entrance through the imperfectly developed skin and mucous membranes. Alexins and other antibodies are markedly deficient in the premature infant, and for this reason it much more readily succumbs to infections which find entrance through the umbilical wound, the skin, mouth, gastrointestinal canal, and, perhaps of even more importance, through the respiratory passages. Premature infants are especially predisposed to all forms of general sepsis, to bronchopneumonia, bronchitis, gastrointestinal disorders and hemorrhagic diseases associated with serious forms of malnutrition.

Very commonly prematurity is produced by some severe constitutional disease in the mother, such as syphilis or tuberculosis. Infants of this type usually suffer from a severe form of hereditary syphilis, or from pronounced malnutritions, which are quite independent of the retardation in development which characterizes uncomplicated prematurity. Infants

who are not only premature but are congenitally weak and malnourished as a result of hereditary disease have much less chance for attaining normal development than has the infant who suffers simply from uncomplicated prematurity.

In addition to the symptoms which have been dwelt upon above, premature infants are markedly underweight, and their birth weight is of great importance from the standpoint of prognosis. Viable premature infants may vary in weight from two and one-half to six pounds. Death almost always occurs if the body weight is less than two and one-half pounds. With the increasing birth weight of the infant the prognosis becomes more favorable. The skin of the premature infant is commonly slightly jaundiced. Its extremities, and in fact the whole surface of its body, feel cool to the touch, and with the feeble and shallow respiratory movements we may have cyanosis, dyspnea, or asphyxia.

**Prognosis.**—The prognosis depends largely upon the rectal temperature and the possibility of producing and maintaining a comparatively normal body temperature under the influence of artificial heat. It depends also, as previously stated, upon the weight of the infant and upon its ability to take and assimilate sufficient food to supply its body wants. Under favorable conditions in private families, where the infant can be at once properly treated without first allowing it to become chilled and to suffer from a low body temperature for a number of hours, the prognosis is good. The majority of cases born after the seventh month and weighing more than three pounds develop into normal, healthy infants. Premature infants who are neglected for the first twelve hours of their lives, and who perhaps during this time are transferred from one institution to another, have greatly diminished chances for living. The prognosis in breast-fed premature infants is vastly better than in those which are fed upon artificial food. When syphilis and other forms of congenital debility are added to the prematurity, the prognosis is for the most part unfavorable.

**Treatment.**—As premature babies very commonly suffer from asphyxia, the earliest treatment of these cases consists in clearing the throat of mucus and other fluids and establishing normal respiratory movements by the resuscitating measures outlined under Asphyxia. Following the establishment of normal respiration, the infant's body is to be cleansed with oil and absorbent cotton and its eyes carefully washed with a saturated solution of boracic acid. It is then to be carefully wrapped in absorbent cotton so that its whole body, except the face, hands and buttocks, is wholly covered; the absorbent cotton should be held in position by gauze bandages. The object of thus covering the infant with a thick layer of absorbent cotton immediately after birth is to prevent the sharp fall in body temperature which may occur at this time. The buttocks are to be protected by separate pieces of cotton so adjusted as to catch the excreta without fouling the entire dressing. The cotton dressing above described is to be changed once in twenty-four hours in a warm room with the infant before an open fire. When the dressing is removed, before another similar dressing is

applied, the infant's body is to be cleansed with cotton and warm olive oil. This form of dressing should continue to take the place of clothing for from one to three weeks, depending upon the stage of immaturity of the infant. As soon as the heat-regulating apparatus of the infant commences to assume normal control of the body temperature, these wrappings may be gradually changed for the clothing ordinarily worn by newly-born infants. The skin, buttocks and mucous membranes of the nose and mouth should be kept clean and free from irritation. The position of the infant should be frequently changed so that no portion of the skin will be subjected to body pressure for any great length of time. This is important since these infants will lie for an indefinite length of time in one position, making no movement and uttering no cry.

Incubator.—The most important part of the treatment is that of maintaining an approximately normal and even temperature of the infant's body by artificial means, without causing it to breathe an overheated impure air. This problem is very difficult of solution; to solve it incubators were introduced and they are now in general use and recommended by all authorities. Only incubators of the most approved type should be used and they require careful supervision by competent attendants night and day. The heat-regulating apparatus of the best of incubators may at times get out of order, and as a result the infant may be exposed to great heat or cold, and such an accident may be fatal to the incubator infant.

The experience of pediatricians has been that an incubator temperature above 85° F. is prejudicial to the welfare of infants and that they thrive best at a temperature of 80° F. With this amount of artificial heat added to that which the infant can manufacture the rectal temperature of the incubator infant should be between 93° and 97° F. Normal temperatures are not to be expected during the first or second weeks of treatment, but if the infant's temperature remains constantly below 90° F. the prognosis is very unfavorable. It is advisable, especially in institutional work, that the air supplying the incubator should come from the outside, so as to have it as pure and as free from microbic contamination as possible. Incubators, when properly constructed and carefully watched throughout the whole of the twenty-four hours, have given good results both in institutional and private practice.

Padded Basket.—My own experience is, in accord with that of many other pediatricians, that the cotton-padded basket is more easily managed and gives as good results as the incubator, especially in private practice. In carrying out this treatment the premature infant, after being cotton-wrapped as above described, is placed in a basket, the inside of which has been previously heavily padded with cotton and covered with gauze, and the space within the basket when thus padded should be at least twice the length of the infant, so that warm water bottles or electric heaters can be so placed as to apply artificial heat to the child's body without coming in direct contact with it. The infant thus covered with warm blankets and its bed warmed by artificial heat is placed in a large, quiet, warm, well-

ventilated room; the temperature of this room should be about 80° F. If the room has an open fireplace this should be utilized during the cold months to assist in warming and ventilating the room, and at least one window in the room should be partially open to let in a stream of pure fresh air. The infant's basket-bed should be placed in the warmest part of the room, away from the draught of windows and doors. All persons, except the nurse and mother, should be excluded from the room, and above all contagions of every kind are to be carefully avoided. All individuals suffering from slight coryzas, colds in the head, or any other catarrhal conditions of the respiratory passages should be excluded from the room. These infants are not only very susceptible to contagions of all kinds, but when once the contagion is started they offer little or no resistance to it.

Fig. 12.—Padded Basket for Treatment of Premature Infants.

A simple rhinitis or an ordinary cold may prove a fatal complication in that it may lead to bronchitis or bronchopneumonia.

The basket, when all the above details of treatment can be carefully carried out, is safer and, as I believe, gives as good results as the incubator. The basket may also be used in institutional work instead of the incubator if the infant can be isolated as above described. This basket treatment, however, in the crowded wards of a city hospital is very unsatisfactory; the incubator with its outside ventilation is preferable under such conditions.

Feeding of Premature Infants.—*Breast Feeding.*—Feeding is of almost as much importance as the treatment above given for maintaining the body temperature and furnishing fresh air. Every premature infant should, if possible, be fed upon breast milk, otherwise its chances for recovery are very greatly diminished. Within the first twenty-four hours the infant requires no food; during this time a little water slightly sweetened with milk sugar may be given; this gives the physician an opportunity to find a wet nurse. The mothers of premature infants are not prepared to furnish breast milk; the milk secretion is not established in most cases

until a week or ten days have elapsed, and then in many instances this result is brought about by the frequent use of a breast pump or by the nursing of another healthy infant. Furthermore, even when the milk secretion of the mother commences to be established it contains for a number of days so much colostrum that it is not a suitable food for a premature infant. A wet nurse should therefore be employed for two or three weeks until the milk secretion of the mother is fully established, and during this time the infant of the wet nurse may be used to develop the milk secretion of the mother and at the same time to keep up the normal supply of breast milk in the wet nurse.

Premature infants are not, as a rule, able to suck; the breast milk must therefore be drawn from the breasts with a breast pump and fed to the infant by means of a pipette or some kind of infant feeder, such as that devised by Breck. If the child of the wet nurse is not allowed to nurse its own mother, the quantity of breast milk will quickly become deficient, but under the stimulating influence of sucking the quantity of milk given by the wet nurse will be quite sufficient to supply both infants. Later the breast milk of the mother may be substituted for that of the wet nurse.

FIG. 13.—BRECK'S FEEDING TUBE.

*Artificial Food.*—In the event that it is absolutely impossible to obtain suitable breast milk an artificial food formula must, of course, be resorted to. During the first three days it is advisable to give a 2 to 4 per cent. solution of milk-sugar, and by the end of the third day closely skimmed milk may be added to the milk-sugar solution, one part of skimmed milk to six parts of a 4 per cent. milk-sugar solution. From day to day the quantity of skimmed milk may be increased until at the end of the week it is taking one part of skimmed milk to three parts of sugar solution. By this time the intestinal canal has been cleared of meconium, and normal intestinal discharges should indicate that the intestinal canal is in a condition to take a modified milk formula containing fat as well as sugar and protein. The infant may then be given 0.5 per cent. fat, 0.3 per cent. protein and 4.00 per cent. sugar. As time goes on the fat and protein content of this food mixture is gradually increased, so that by the end of the third week the infant may be taking a 1.00 per cent. fat, 0.75 per cent. protein and 5.00 per cent. sugar mixture. As the infant thrives the protein and fat percentages in this formula are to be slowly increased according to the rules outlined in the chapter on Artificial Feeding.

*Quantity of Food.*—As both the breast milk and the modified cow's milk are fed to these infants with some kind of a feeding tube, the quantity of food taken can be accurately measured. After the second or third day, when the feeding with breast milk is begun, the infant should have from four to seven ounces of milk in twenty-four hours. The quantity given

will depend upon the weight of the infant. The four ounces is suitable for an infant weighing between two and three pounds; the seven ounces for an infant between five and six pounds. Day by day as the infant grows older the quantity of breast milk is increased, so that by the end of the second week the infant which began with four ounces will be taking fourteen or fifteen ounces in twenty-four hours, and the infant which began with seven ounces will be taking seventeen or eighteen ounces in twenty-four hours. The same quantities of modified cow's milk may be given to those infants who are unfortunate enough to be deprived of breast milk. The interval between the feedings should, in the beginning, be one and one-half hours. At the end of two weeks this interval should be prolonged to two hours. This will make the individual feedings vary from one-half an ounce in the very small premature infant to one ounce or one and one-half ounces in the large premature infant. As soon as the infant is strong enough it should be put to the breast. In many cases it may be possible to obtain a small but insufficient quantity of breast milk. In such cases mixed feeding should be resorted to, and this mixed feeding should be followed out according to the method carefully detailed in the paragraph on Mixed Feeding in the section on Artificial Feeding of Infants. All of the breast milk that can be obtained should be given at each feeding and this is to be supplemented by a modified milk formula given at the same time and in sufficient quantity to make up the deficiency.

Premature infants which thrive properly should develop into normal, sturdy children, leaving no trace of weakness as a result of their prematurity. In the beginning these children, like normal infants, lose slightly in weight, but after a week or ten days they should have regained their birth weight, and thereafter should continue to slowly increase in weight. In the beginning a gain of two to three ounces per week is considered satisfactory, but after five or six weeks, when they are strong enough to nurse the breast and to take larger quantities of milk, their gain in weight should become more rapid.

## CHAPTER VI

### DISEASES OF THE NEW-BORN

### ASPHYXIA NEONATORUM

**Etiology.**—Asphyxia is due to deficient oxygenation of the blood and the resulting symptoms are in part produced by the poisonous action on the nerve centers of carbon dioxide. As the infant during intrauterine life is dependent upon the placenta for its supply of oxygenated blood it may be asphyxiated by anything that interferes with the placental circulation. The most common cause of this condition is pressure or twisting of the umbilical cord during labor. The cord may be prolapsed or it may be

wrapped around the neck or some other portion of the infant's body in such a manner that, during delivery, especially if it be prolonged, the circulation between the placenta and the infant is cut off and asphyxiation results. This is more liable to occur during breech presentations and during protracted labor following the premature discharge of the liquor amnii; in these conditions the cord is so firmly pressed against the body of the child by the strong uterine contractions that circulation through it is impeded or entirely obstructed. Asphyxia may also be produced by cerebral hemorrhage, by defective development, by the premature detachment of the placenta and by the death or serious illness of the mother during labor. If the child is asphyxiated before labor begins it is stillborn and the extent of the maceration of its skin and the general appearance of the dead fetus may give some idea of the length of time it has been dead.

Asphyxiation may also occur after birth in premature and malnourished infants as a result of defective development of the muscular and nervous mechanisms which preside over the respiratory movements. In this type the infant, which has been kept alive by the placental circulation during intrauterine life, has not sufficient vitality to establish normal respiratory movements after birth. Fortunately these hopeless cases are very uncommon. Criminal neglect of the infant just after birth may allow it to lie face downward in the blood and mucus which has been discharged during labor, and in this manner it may become asphyxiated.

**Symptomatology.**—In those cases where asphyxia occurs during labor the carbon dioxide poisoning and the air hunger bring about premature inspiratory efforts, and, as a result, mucus and other secretions may be drawn into the respiratory passages and by strangulation increase the existing asphyxia.

The symptom groups which characterize the mild and severe types of asphyxia are somewhat distinct. The mild form is spoken of as asphyxia livida. In this condition the skin is blue and the mucous membranes are a dark purple color. The infant lies more or less motionless, but is not limp or apparently lifeless; its muscles are not relaxed, its reflexes are commonly present, its pupils are not dilated and the action of its heart can be distinctly heard and commonly felt by placing the finger over the location of the apex beat. The infant, however, does not cry, and its respiratory movements are irregular, shallow or gasping.

In the severe form known as asphyxia pallida (Runge) the child at first sight is apparently dead. It has a pale, pasty, cadaverous look about its face; its lips are dark blue, its body and extremities are cold and there is a general lack of tone or flaccid condition of all the muscles. As the child is lifted its body seems limp and lifeless, reflexes are absent, pupils are dilated, there are no efforts at respiratory movements, and the only evidence of life is to be found in the feeble heart beat, which can be heard but not felt. In some of these cases a few gasping efforts at inspiration may be made, but the mucus which has accumulated in the throat and upper respiratory passages prevents the entrance of air into the lungs. It

is evident from the above description that there is no clear line of demarcation between asphyxia livida and asphyxia pallida. The livid and pale forms of this disease represent but different grades of severity. One may meet cases of asphyxia so mild that slight cyanosis and irregularity in breathing are the only symptoms, and again the case may be so severe that all efforts at resuscitation fail, and the clinical picture of asphyxia pallida is aggravated until the cessation of the heart beat announces the death of the infant. Between these two extremes we may have every grade of severity.

**Diagnosis.**—It is important to remember that cerebral hemorrhage occurring during labor may produce a symptom group closely resembling asphyxia. Perhaps it might be more accurate to say that asphyxia is a part of the symptom group in many of the cases of cerebral hemorrhage. It is important, therefore, in all cases of asphyxia neonatorum to withhold the ultimate prognosis until it can be determined whether or not there is a coexisting cerebral hemorrhage; this can in most instances only be decided by the subsequent history. In asphyxia, when respiration has been established, the improvement is very marked, but while the child is weak there may be a tendency to a slight return of the asphyxia in the first twenty-four or thirty-six hours, yet under careful nursing satisfactory convalescence is soon established. This is not true of cerebral hemorrhage severe enough to produce asphyxia. In these cases we are likely to have localized or even slight general convulsions recurring over a number of days, and the infant during this time remains in a dull and stupid condition. Thereafter convalescence, as compared with that of uncomplicated asphyxia, is very slow. It should be remembered that in these cases of cerebral hemorrhage one may *not* have for weeks and months the characteristic symptoms of spastic palsy, so that the absence of this palsy in the newly-born infant suffering from asphyxia does not exclude cerebral hemorrhage.

**Prognosis.**—The prognosis depends largely upon the character of the asphyxia and the treatment instituted. If well marked it is always a grave symptom. In general terms, however, one may say that the prognosis in asphyxia livida is good if the cases are properly treated, and that the prognosis in asphyxia pallida, while bad, is not always fatal. In cases due to cerebral hemorrhage the prognosis as to life is unfortunately good. I say unfortunately, since nearly all of these cases are hopelessly defective in their mental development (Jacobi).

**Prophylaxis.**—The preventive treatment of asphyxia is largely obstetrical. Breech presentations and tedious labors, especially in cases where the liquor amnii has been discharged prematurely, should be terminated as rapidly as possible. In cases of this kind, where asphyxia may be expected to result, the infant immediately after birth should receive prompt and skilful attention. The mucus and other foreign matter in its throat should be quickly wiped out with a moist cloth covering the finger, and it should be held up by the legs, head downward, and gently shaken, as this procedure facilitates the removal of the inspired fluids and stimulates by congestion

the respiratory centers. Slapping the body of the infant with a cool rag or *dipping it alternately into a bucket of warm and cool water* may reflexly stimulate respiratory movements. This may be done three or four times in a minute until the infant begins to cry and more or less normal respiratory movements are established. This treatment when promptly administered will prevent many cases of asphyxia.

**Treatment.**—When the infant is born asphyxiated the object first sought is to clear the respiratory passages of inspired mucus and liquor amnii. This is accomplished, as above stated, by holding the infant head downward, shaking it, and at the same time, with a gauze-wrapped finger, removing the mucus and other fluids from the throat. If evidences of strangulation still exist a small, soft, rubber catheter, cut off at the end, should be introduced into the opening of the larynx and the fluids removed by suction or aspiration. In emergency cases of this kind there is no time for the operator to provide himself with an instrument especially devised for this purpose, and valuable time should not be lost in preparing an instrument which will protect the mouth of the operator from these inspired fluids which are being drawn from the respiratory passages of the infant. These manipulations having been made, as quickly as possible, to clear the respiratory passages of fluid, the child is placed upon a bed face upward and a piece of gauze thrown over its mouth; the physician then, after closing the nose of the child with one hand and making firm pressure over the stomach with the other, places his lips to that of the infant and blows air from his own lungs into those of the infant, and as he thus inflates its lungs he can see the movements of the chest walls; the air is expelled from the lungs by pressing upon the chest wall. This method of lung inflation may be repeated every few minutes until the infant begins to cry or makes efforts at normal respiratory movements; artificial respiration should then be resorted to.

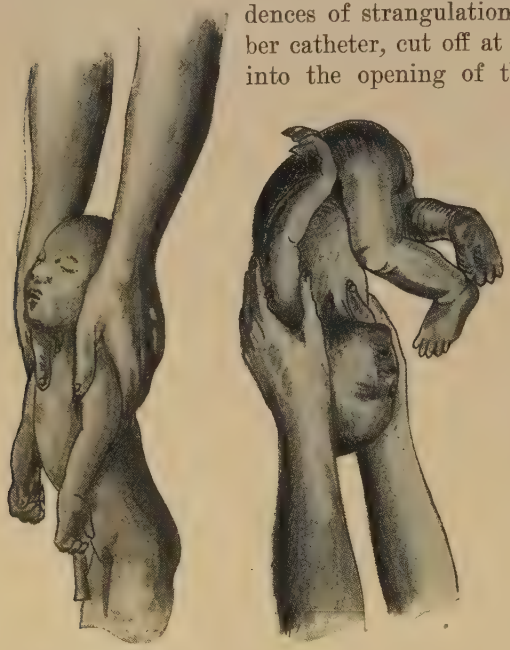


Fig. 14.—Schultze's Method of Artificial Respiration. (After Edgar.)

Artificial Respiration.—Schultze's method, which is recommended by all writers upon this subject, is the most valuable and is described as follows: The physician, standing, grasps the infant with both hands, his

palms resting upon the child's shoulders, his thumbs extending over the anterior surface of the chest near the axilla, his fingers spreading out over the scapulæ, and the infant's head, resting between his arms, is supported by his wrists. The infant, firmly grasped in this manner, is swung upward above the operator's head; in doing this its body is bent forward, its abdominal viscera pressed upward against the diaphragm and expiration is thereby accomplished. The child's body is now swung backward in the same circle until its body hangs downward with its spine bent slightly backward; with this movement the diaphragm sinks and inspiration is accomplished. This operation is repeated some ten or fifteen times a minute until normal respiratory movements begin to be established. In employing this method unnecessary chilling of the infant's body is to be avoided as much as possible.

Other methods of artificial respiration are recommended. A modification of the Schultze method described by Dew consists in holding the infant back downward in such a way that its body may be bent in much the same manner as in the swinging movements above described. The body of the child is alternately flexed and extended so as to push the diaphragm upward and pull it downward, thus producing expiration and inspiration. These manipulations may be made, as Ela recommends, while the infant is in a warm bath, temperature 100° to 105° F. The combination of warm bath and artificial respiration is especially to be recommended in asphyxia pallida. Artificial respiration may also be carried on in young infants as it is in older children by placing the child, face upward, in a prone position with its shoulders slightly elevated above the rest of its body, and then gradually lifting the arms high above the head in a line with the body and again bringing them down, at the same time making compression downward and inward against the whole anterior and lateral surfaces of the chest wall.

Following its resuscitation and the establishment of normal respiration the infant must be carefully watched for twenty-four or thirty-six hours, and in the event that signs of asphyxia begin to recur it must again be subjected to one or the other methods above detailed for bringing about normal breathing. The inhalation of oxygen is sometimes of benefit in these recurrent cases. Strychnia, 1/400 or 1/500 of a grain, given hypodermically, has also been recommended in these cases. As some of the severe cases are unable to nurse for three or four days, it may be necessary to feed them with a medicine dropper and to give them occasionally a few drops of whiskey well diluted.

## CONGENITAL ATELECTASIS

The lungs of the infant at birth are collapsed, contain no air, or, in other words, are in a state of congenital atelectasis. With the first inspiratory efforts the lung commences to expand. This is a gradual process and a number of days elapse before the entire lung is inflated. If any portion

of the lung fails to expand, it remains in its fetal condition of collapse or congenital atelectasis. It is, moreover, evident that the extent of this condition may vary greatly from a slight and scattered atelectasis, which results simply from delay in the inflation of the lung, to an atelectasis so extensive that the whole of one lung may be involved or such extensive regions of both lungs that life under these conditions is impossible, and the infant dies from asphyxia.

**Etiology.**—All of the causes which predispose to asphyxia neonatorum are also important factors in the production of atelectasis. It occurs in premature infants, in feeble, malnourished infants, in those suffering from congenital syphilis, and in infants suffering from cerebral hemorrhages and other birth injuries. Inspired fluids, such as mucus and liquor amnii, which may be sucked into the bronchial tree with the first inspiratory efforts, may obstruct the smaller bronchi and prevent the inflation of the portions of the lungs to which they carry air. Obstructive atelectasis may also occur after normal respiratory movements have apparently been established. In such cases portions of the lung which have been inflated again return to their atelectatic condition, thus producing a form of acquired atelectasis. These cases of acquired atelectasis occurring in the new-born are, as a rule, due to the plugging of the bronchial tubes with mucus or with inflammatory products. In this chapter, the acquired atelectasis which occurs in *older infants* as the result of bronchitis, bronchopneumonia, pleurisy and rickets, will not be considered.

**Symptomatology.**—The symptoms are commonly associated with, and practically cannot be separated from, those of asphyxia neonatorum, or perhaps one might more clearly express this relationship by saying that a greater or less degree of asphyxia is a constant symptom of atelectasis. When the atelectasis is so slight as not to produce any evidences of asphyxia whatever, then it cannot, as a rule, be discovered by other physical signs and symptoms. The respiratory movements in atelectasis are defective. They are usually irregular and shallow. Sometimes long pauses occur, followed by a gasping respiration, with the resumption for a time of irregular and superficial respirations. The more pronounced these symptoms the more aggravated the case. In the milder cases respiratory action is but slightly interfered with. In the most severe cases complete asphyxia occurs.

These infants are, as a rule, feeble, somnolent and ominously quiet, not demanding, and usually not taking, sufficient nourishment. The temperature is subnormal, the face pale and comparatively expressionless. On deep inspiration, which may be brought about by slapping the child with the hand, or applying cool water to its chest, one may hear, at the base of the lungs, crepitant or subcrepitant râles. Where the atelectasis is severe, and more marked on one side than on the other, lack of symmetry in the respiratory movements of the chest wall may be observed. In those cases which live for weeks without the disappearance of all the symptoms of atelectasis it is possible or even probable that an afebrile, insidious broncho-

pneumonia may develop at any time. In such cases the physical signs of a bronchopneumonia are commingled with those of atelectasis.

**Treatment.**—The somnolence and shallow, irregular breathing of these children are to be combated by much the same measures recommended for the treatment of mild cases of asphyxia. From six to eight times during every twenty-four hours these infants should be thoroughly aroused and made to take a number of deep inspirations by dipping them alternately into warm and cold baths, as recommended in asphyxia. In milder cases, or as improvement goes on, the same result may be accomplished by shaking and slapping the child and by wiping its face and chest with a cloth that has been dipped in cool water. As these infants commonly have a subnormal temperature it is necessary that they should be kept in a comparatively warm room, and sometimes, in addition to this, artificial heat in the form of hot water bottles placed in the bed near the feet of the infant is necessary. The fulfillment of these conditions makes it almost impossible during the cold winter months to give them the amount of fresh air they require; the windows cannot be opened and they cannot be subjected to the fresh air treatment recommended in pneumonia; for this reason oxygen inhalations are of the very greatest importance. Perhaps most important of all is the feeding of these infants; they are, as a rule, too feeble and breathless to nurse milk from the breast, and yet breast milk is almost absolutely necessary to their proper nutrition. Feeding upon artificial food is not to be thought of unless it be absolutely impossible to obtain breast milk. Until the infant is able to nurse the breast milk should be drawn with a breast pump and fed with an ordinary medicine dropper. In such cases it is necessary, as a rule, to employ a wet nurse, or to secure the services of another infant to nurse the breast and develop the milk secretion of the mother.

## CHAPTER VII

### DISEASES OF THE NEW-BORN (*Continued*)

### SEPTIC INFECTION IN THE NEW-BORN

**Etiology.**—This is an infection produced by pus-forming organisms, such as the streptococcus pyogenes, staphylococcus pyogenes aureus and albus, colon bacillus, pneumococcus, bacillus pyocyaneous, and occasionally by other microorganisms, such as the gonococcus, bacillus enteritidis and bacillus of Friedlander. When these microorganisms find their way into the blood and internal organs of the infant a condition of sepsis is nearly always produced. Localized lesions of the umbilicus, skin, vagina, eye, mouth, etc., may be produced by these microorganisms without a general sepsis supervening. These localized conditions, unless they be associated with a bacteremia or with inflammation of internal organs produced by the

same microorganisms, are not to be considered under the term sepsis as used in this chapter.

Newly-born infants are very prone to septic infection. In the first place, because as compared with older infants and children the portals of entrance for septic organisms are more numerous and more open, and in the second place because at this age there is comparatively little natural resistance to these microorganisms when they have once found an entrance into the blood or other deeper tissues. This feeble resistance is perhaps closely associated with the undeveloped condition of lymphatic structures and with the comparative deficiency in the blood at this age of antibodies and other protective agents with which nature in older children and in adult life very successfully fights bacterial invasion. Breast-fed babies offer more resistance to septic infection than do those fed upon artificial food. This is perhaps because they derive from the milk of the mother the antibodies to which her partial immunity is due. Septic infection is more common and runs a more severe course in premature infants or those who are congenitally weak and in those suffering from profound malnutrition due to lack of proper food, hereditary disease or other causes.

**Portals of Entry.**—Infection commonly occurs through the umbilicus. In a large percentage of the cases the portal of entry cannot be discovered, but there is little doubt that many of these are umbilical in origin. It is perhaps wise to assume umbilical infection in all doubtful cases, even in those in which the umbilicus appears normal. The thrombi which form in the ligated umbilical veins may easily become infected by the pyogenic organisms associated with the necrotic disintegration of the stump of the cord. Under such infection these thrombi break down into purulent material, phlebitis results and the septic matter in the umbilical vein finds its way into the general circulation. In this way a general septicemia is produced, and emboli may be carried into almost any organ in the body, producing localized septic processes. The liver, lungs, intestinal canal and membranes of the brain may thus become infected. The liver, in fact, bears the brunt of the septic onslaught in all cases of septic infection originating at the umbilicus, as the blood from this region is carried through the liver into the general circulation. Septic infection may find entrance through abrasions, fissures or ulcerations of the skin or mucous membranes. It may be introduced into the mouth or nose by unclean fingers or dirty bath water. The lungs and intestinal canal may be portals of entrance for a general septic infection. In these cases it is believed that infected mucus and liquor amnii reach the lungs during the first inspiratory movements or enter the intestinal canal in the early efforts of deglutition. Septic infection may occur through the ear and much more rarely through the eye and genital tract.

**Source of the Infection.**—The infection, as above noted, may have its origin in the sloughing of the stump of the umbilical cord, but it is believed that in the great majority of the cases, even those which have their portal of entrance through the umbilicus, the infection comes from without

and the umbilical wound is inoculated, in some way, with septic organisms other than those engaged in the normal necrotic process incident to the removal of the stump of the cord. This inoculation may occur not only to the umbilical wound, but to any of the other portals of entrance previously mentioned. The common sources of infection are the vaginal discharges of the mother, unclean hands of the nurse or physician, dirty clothing, contaminated air or impure breast milk and dirty bath water. In short, infection may result from any agent which carries septic microorganisms to the portals of entrance which happen to be open in the individual infant.

Septic infection may also be transferred directly through the placental circulation to the unborn fetus by a septic mother. This method of transmission is comparatively rare and is of little etiological importance in the disease under discussion. A large number of other microorganisms, such as the tubercle, the typhoid, the cholera, and the influenza bacillus, and the specific contagion of measles, scarlet fever, pneumonia, and other acute infections, may occasionally be transferred from the mother through the placental circulation to the unborn infant, but infections such as these have been elsewhere considered and have no bearing on the disease under discussion.

**Symptomatology.**—The clinical syndromes presented by septic infections in the new-born vary greatly and the symptoms which announce the onset of this condition depend largely upon the portal through which the septic organisms have entered. In umbilical infection there is commonly a sharp elevation of temperature followed by a few days of septic fever and thereafter the temperature may be normal or subnormal. Jaundice is a common and early symptom. The liver is generally enlarged and the umbilicus is usually inflamed and contains pus, which may be seen filling the umbilical depression, or may be caused to ooze out of the patulous umbilicus on pressure. It should be remembered, however, that a normal umbilicus does not positively exclude umbilical infection. Suppuration may be going on in the thrombi filling the umbilical vein without external evidence of such condition. In this form of sepsis abdominal tenderness, distention and general peritonitis usually develop.

Where the infection enters through the skin or mucous membrane there is, as a rule, some ulceration or abrasion which indicates the point of entrance of the poison. In such cases erysipelatous eruptions, pemphigus, pressure sores, deep ulcerations of the skin, furuncles, edema, sclerema, or gangrene may be observed.

Where the infection enters through the mouth ulcerations of the tonsils or of the mucous membrane of the mouth or nose may be observed. In some instances the mucous membrane is fissured, dry, and patches of thrush or small aphthous ulcers may be scattered over it.

Where the lungs are the portal of entrance there is associated with the general sepsis an early bronchitis, pneumonia, bronchopneumonia, pleuropneumonia, or empyema, so that the general sepsis is largely obscured by these local manifestations.

When the gastrointestinal canal is the portal of entrance the symptoms of general sepsis are largely obscured by the gastroenteric infection and acute enteritis, which are early manifestations.

When the ear is the portal of entrance symptoms of meningeal irritation followed by meningitis and often by paralysis of the face or other muscles are early symptoms.

Individual Symptoms.—As previously noted, the common portal of entrance is the umbilicus, and therefore the syndrome above noted as announcing this disease when it comes from umbilical infection is far and away the most common mode of onset in septic infections of the new-born. This gives prominence to jaundice as a symptom of this disease. Jaundice, however, is not always present. In many cases, as the disease progresses, the face of the infant presents a gray and sickly pallor; in other cases a marked cyanosis is present. Purpura occurring as a fine petechial rash or as large dark-blue spots scattered over the body is also a common symptom of advanced sepsis.

The fever is very irregular and misleading. For a few days following the onset it may be septic in character, reaching 104° or 105° F. at some time during the day, and falling below normal at another. In a few days, however, associated with the *extreme exhaustion,* which is characteristic of this disease, the temperature may fall and remain below normal with perhaps but slight variations.

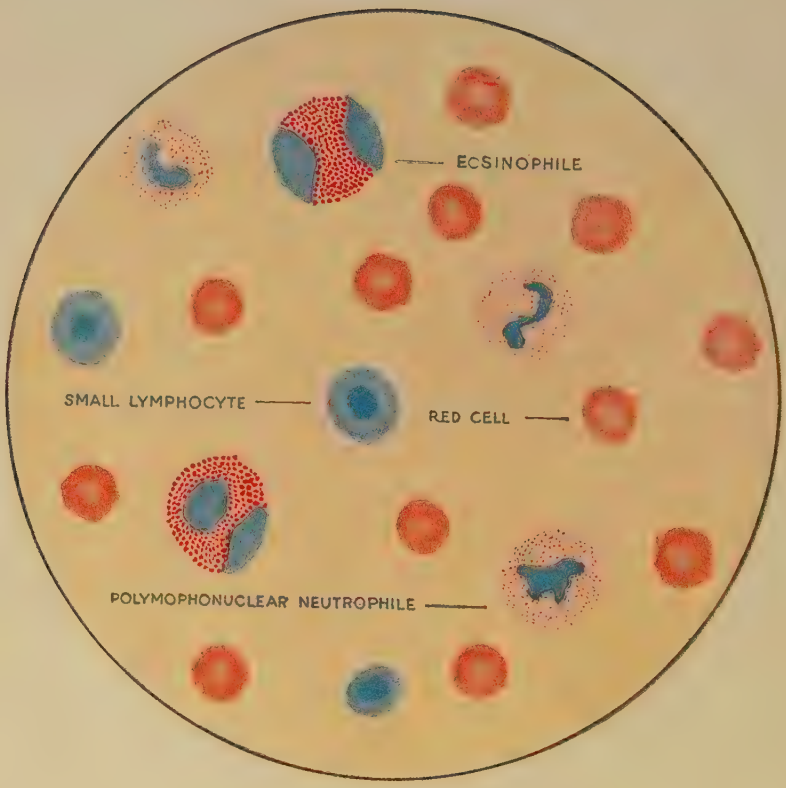
The hemorrhagic symptoms of sepsis are of great importance. Besides the purpura above noted, hemorrhages may occur from nasal, buccal, intestinal and other mucous membranes. Under the heading Hemorrhages in the New-Born the hemorrhagic syndromes of sepsis, which present more or less distinct clinical pictures, are described.

Nervous symptoms of sepsis vary greatly in different cases. As a rule, extreme prostration is associated with apathy and stupor leading up to profound coma. In other instances, especially where the meninges and cerebral centers are involved, there are sleeplessness, extreme irritability, muscular twitchings, localized paralyses and finally convulsions.

In addition to the widely varying symptom group above detailed, we may have, as symptoms of sepsis in the new-born, purulent arthritis, osteomyelitis, pericarditis and, very rarely, endocarditis and nephritis. Albuminuria, associated with occasional hyalin and granular casts, is a very common finding. Purulent vaginitis and conjunctivitis may occur.

**Diagnosis.**—The diagnosis of septic infection in the new-born is oftentimes extremely difficult, since there is no clearly defined clinical syndrome which can be relied upon to definitely indicate this disease. The physician, however, should always keep in mind the fact that severe and dangerous gastrointestinal, pulmonary and meningeal symptoms occurring at this time of life are strongly indicative of sepsis. The cases most difficult of diagnosis are those presenting the symptoms of pneumonia, gastroenteritis and meningitis. In the majority of cases there is external evidence either at the umbilicus or on the skin or mucous membranes, which indicates that the

PLATE II.




Blood Picture in Dermatitis Exfoliativa.
(Drawn by Dr. A. E. Osmond).

infant may be suffering from septic infection. The symptoms which especially call attention to this condition are great and unexplained prostration, associated with jaundice, septic temperature and hemorrhages of the character above noted. The blood examination may show a marked leukocytosis. Blood cultures which might demonstrate positively not only the existence of a general septic infection, but the causative organism as well, cannot be resorted to in infants of this age as a routine method of diagnosis.

**Prognosis.**—This is very grave; nearly all severe cases rapidly succumb. Death may occur within a few days, or it may be postponed for a week or more. Many of the milder cases, especially those having their origin in umbilical infection, recover. The prognosis is altogether unfavorable in the pulmonary, gastrointestinal and meningeal cases.

## DERMATITIS EXFOLIATIVA

**Symptomatology.**—This interesting and comparatively rare syndrome described by Ritter was believed by him to be a manifestation of septic

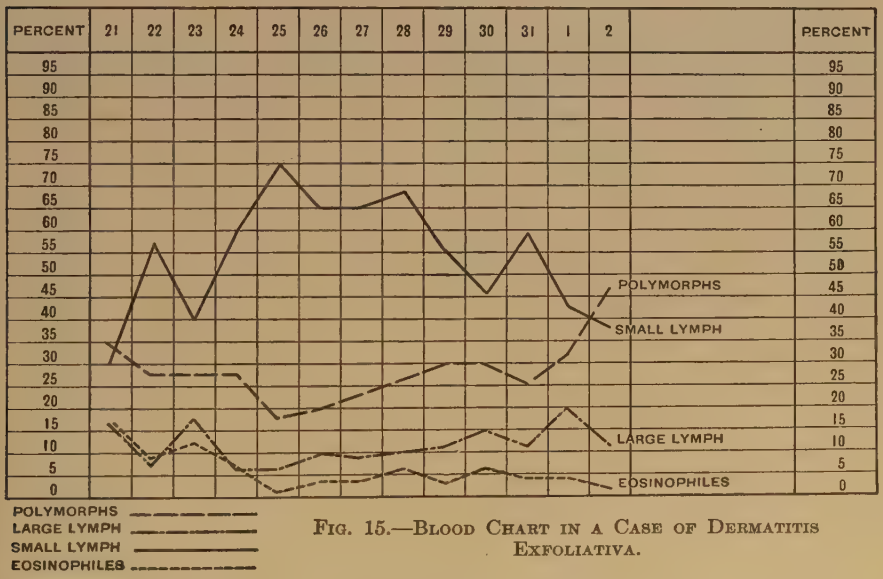



FIG. 15.—BLOOD CHART IN A CASE OF DERMATITIS EXFOLIATIVA.

infection in the new-born. It makes its appearance between the first and the third week of life. In the beginning the skin of almost the entire body begins to show hyperemia with more or less swelling and superficial inflammation, and fissures appear at the angles of the mouth. This latter symptom is important and quite characteristic when taken in connection with the general skin eruption. As the dermatitis proceeds the skin becomes thicker, more edematous and covered with large flaky scales of epithelium, which are partly detached and which give to the whole body a characteristic scaly appearance. These scales may be removed in large flakes, exposing the

congested skin beneath. If recovery occurs, as it does in some instances, this flakiness of the skin gradually diminishes and in the course of a few weeks entirely disappears. In the great majority of cases, however, the disease is associated with symptoms of general sepsis and the prognosis in these cases is bad. In a case recently observed by me the dermatitis exfoliativa occurred as a symptom of general sepsis following umbilical infection, and the blood picture in this case was interesting and remarkable. This blood picture, so far as the large number of eosinophiles was concerned, was perhaps due to the skin lesion. The marked leukocytosis was perhaps a symptom of the general sepsis. The blood picture in this case is here presented.

**Treatment.**—There is perhaps no treatment which materially influences the course of general sepsis in the new-born. Unguentum Credé, as an inunction, may be thoroughly rubbed through the skin twice in every twenty-four hours; the technique for its administration is given in detail in the chapter on Scarlet Fever. Any disease of the umbilicus or of the skin or mucous membranes of the mouth should have appropriate treatment. In umbilical infection, the wound is to be washed out with a 1 to 4 solution of peroxid of hydrogen or a 1 to 1,000 solution of bichlorid of mercury. Following the use of these disinfectants the part is to be carefully dried and covered with aristol, or some other antiseptic powder, which is held in place by a gauze pad and proper bandages. External wounds and ulcers of the skin should be treated in much the same way. The infant, if not able to nurse, is to be fed with breast milk from a medicine dropper, and given from ten to fifteen drops of whiskey in two teaspoonfuls of water every three hours.

The *prophylactic treatment* of this condition is of the greatest importance and is altogether in the hands of the obstetrician. The infant should be delivered and handled with clean hands, and immediately following its birth should be bathed in clean, warm water, great care being taken to protect the eyes, the nose and the mouth of the child. If the obstetrician knows of vaginal conditions in the mother which predispose the unborn child to sepsis, even greater care must be exercised to prevent infection during labor; saline vaginal douches may be of value in such cases. Following the tying of the cord the umbilical region must from time to time be carefully inspected, and if evidences of acute inflammation develop, or if pus appears, the antiseptic treatment above noted for the relief of this condition should be resorted to.

Infants should not be allowed to nurse breasts with infected fissures or in which there has already developed an abscess or localized inflammatory swelling.

## ERYSIPELAS

The specific organism of this disease has not been isolated. It is believed to be an inflammation of the skin and mucous membranes produced by septic cocci, and the characteristic redness and edema are perhaps due to

the location of the septic inflammation in the skin and subcutaneous tissues. It is produced by septic virus coming in contact with an open wound, or with some abrasion of the skin or mucous membrane. This inoculation most commonly occurs during the first two weeks of life through the umbilical wound, but it may occur through abrasions of the skin and mucous membranes about the nose, genitalia and other portions of the body.

**Symptomatology.**—Erysipelas manifests itself by a well-marked redness and swelling of the skin, which in the new-born commonly occur about the umbilicus. Because of the feeble resistance of the infant to this disease the inflammation spreads rather rapidly through the skin and subcutaneous tissues around the umbilicus and over the lower portion of the abdomen and may extend to the lower extremities. When the infection starts at the umbilicus the inflammation spreads downward, although in some instances it may involve the skin of the chest, face and upper extremities. When the erysipelas begins at the angle of the nose or some portion of the face it spreads rapidly over the face and head of the infant and thence downward, involving the skin of the chest and other portions of the body. Facial erysipelas in the infant, unlike that in the adult, is not, as a rule, limited to the face and head.

Erysipelas in the infant frequently results in a general sepsis, producing peritonitis, pneumonia and other septic infections of internal organs, and the subcutaneous tissues, more commonly than in the adult, are involved in a phlegmonous inflammation. The constitutional symptoms are usually severe. High fever, marked prostration, intestinal disorders, somnolence and even convulsions may occur.

**Prognosis.**—The younger the child the more unfavorable the prognosis. Umbilical infections are commonly more serious than those occurring in other parts of the body. In children over one year of age the prognosis is usually good, the disease running its course and terminating in recovery in much the same manner as it does in the adult; during the first weeks of life it is a very fatal disease. The great majority of these cases die from general sepsis.

**Treatment.**—The treatment of erysipelas in the young infant is very unsatisfactory. Every effort should be made to nourish the child with breast milk. In view of the fact that these cases are commonly complicated by intestinal disturbances the infant should from the beginning be fed as though it had gastroenteritis, and medicines, such as the tincture of the chlorid of iron, which may produce gastric disturbance, should not be given. Whiskey, well diluted, may be used throughout the disease. Ichthyol ointment, one drachm to the ounce of lanolin, is almost universally recommended as a local application. It relieves the irritation of the skin and thereby diminishes the nervous irritability of the child, but it does not have any specific influence in controlling or checking the spread of the inflammation. Antistreptococcic serum in from 2 to 5 c.c. doses, given hypodermically at intervals of six or eight hours, is of value in some cases.

Unguentum Credé should be given by inunction in all cases. This ointment should be thoroughly rubbed into the skin of the unaffected portions of the body twice in twenty-four hours. I believe that this treatment may be of material advantage in controlling the sepsis in many cases.

## HEMORRHAGES IN THE NEW-BORN

There are a number of clinical syndromes, occurring in the new-born, in which hemorrhage is the most striking symptom. All of these syndromes are believed to be due to some kind of infection, but they differ somewhat from the clinical pictures of septic infection just described. It is to be remembered, however, that ordinary sepsis is the most common cause of spontaneous hemorrhage in infants just after birth, and it is also to be remembered that hemorrhages may very rarely occur from congenital hemophilia and from unknown causes which are apparently not septic in their origin. In all of these conditions the hemorrhages are due primarily to impaired blood coagulation; the exciting traumas are insignificant.

### EPIDEMIC HEMOGLOBINURIA

(*Winckel's Disease*)

Epidemic hemoglobinuria, which is elsewhere noted, is characterized by a well-marked hemoglobinuria, great depression and icterus. The urine is dark red in color and contains besides hemoglobin a small amount of albumin with occasional casts.

### ACUTE FATTY DEGENERATION OF THE NEW-BORN

(*Buhl's Disease*)

Acute fatty degeneration of the new-born is a rare septic syndrome, in which there may be hemorrhages from the umbilicus or from the gastrointestinal, conjunctival and buccal mucous membranes, or petechial hemorrhages may occur beneath the skin. Asphyxia, icterus, and edema are commonly present; the spleen and liver are enlarged. There is early and profound prostration ending in death within one or two weeks. In this condition the heart, liver and kidneys undergo a rapid fatty degeneration, and hemorrhages may occur in these organs.

### MELENA NEONATORUM

Melena neonatorum is a term used to cover a septic syndrome, the characteristic symptom of which is bleeding from the gastrointestinal canal. In these cases blood is discharged from the rectum and is ejected from the stomach. The hemorrhage occurs during the first few days of life and the first symptom commonly noticed is the discharge from the bowels of dark-red or black material, which on examination is found to be blood. In the later discharges the blood, not remaining in the intestinal canal so long, is not so dark in color. In those cases where the bleeding continues vomiting of blood occurs and the blood continues to be discharged through the rectum.

The child becomes more and more prostrated, its pallor deepens, the heart sounds become more and more feeble, and death occurs from exhaustion. There are, however, a considerable number of cases in which there is but little or no recurrence of the hemorrhage after birth. In these cases the infant may discharge by the rectum brown or black discharges mixed with mucus and fecal matter, an examination of which shows that this discoloration of the fecal discharges is due to blood. These cases may run a benign course, the dark-brown discharges colored with blood may continue from four to five days, gradually diminishing in frequency and gradually losing their dark color, and thereafter the child may show no evidence of disease of any kind except perhaps a slight intestinal indigestion, which may continue for some weeks. It is very questionable whether these benign cases are septic in origin; it is much more probable that the hemorrhages are due to injuries received during birth. Even severe cases of intestinal hemorrhage, which have continued for more than a week, may recover. The bleeding in some instances stops spontaneously, and the infant makes a slow but satisfactory recovery, and thereafter it may never manifest any hemorrhagic tendencies or constitutional taints to explain the symptoms from which it suffered during the first days of life. A number of these cases have come under my observation.

### CONGENITAL SYPHILIS

Congenital syphilis may have among its earliest manifestations hemorrhages from the nose, mouth and other mucous membranes. In fact, when hemorrhage occurs during the first days of life from the nose of an infant it is well to suspect and look for other symptoms of congenital syphilis. Pronounced and troublesome umbilical hemorrhages are nearly always septic in their origin.

### PROGNOSIS OF HEMORRHAGE

This depends upon the location, the severity and the cause of the hemorrhage. In Winckel's and Buhl's diseases the prognosis is bad. In ordinary sepsis of umbilical origin hemorrhage is an unfavorable symptom and commonly means a fatal termination. In hemorrhages from the nose or buccal mucous membrane due to syphilis the hemorrhage can usually be controlled by the local application of a solution of adrenalin, and the prognosis will depend upon the severity and extent of other syphilitic lesions. In melena neonatorum (hemorrhage from the gastrointestinal canal) the prognosis is very grave and will depend in part upon the presence of other symptoms of sepsis.

### TREATMENT OF HEMORRHAGE

Hemorrhages from the mouth and nose may be controlled by the local application of adrenalin solution, and gastrointestinal hemorrhages may sometimes be modified or controlled by the internal administration of adrenalin in 2 or 3-grain doses given at intervals of two or three hours. Ten per cent. sterile gelatin solution injected subcutaneously (15 c. c.) has

also been strongly recommended in these cases. These infants should be kept as quiet as possible and no foods or fluids given except water until the hemorrhage is controlled; then small quantities of breast milk at four-hour intervals may be allowed. Cathartics are to be avoided and enemata used only when it is necessary to evacuate the bowels.

Hemorrhagic diseases in the new-born have been successfully treated by direct transfusion of human blood (Carrel); the technique of this operation, however, is difficult and for this reason it is not always practicable. They have also been successfully treated by injections of the normal serum of human blood. This method was introduced by J. E. Welsh and has been used by many other observers. The blood serum is obtained from human blood withdrawn and cared for under sterile conditions. Ten to thirty c. c. should be injected subcutaneously two or three times a day as long as the bleeding continues, and the same treatment should be resumed if the hemorrhage returns. Following both the transfusion of human blood and the subcutaneous injections of human blood serum, improvement begins at once and the hemorrhage is usually controlled within one or two days. In the great majority of these cases a permanent cure results; in a few, however, the benefit derived is of short duration; the hemorrhage returns and the disease goes on to a fatal termination. These methods of treatment should therefore be used in all severe cases of hemorrhagic disease in the new-born.

## DISEASES OF THE UMBILICUS

After ligation the stump of the umbilical cord undergoes mummification and comes off about the fifth or the sixth day. In premature and congenitally weak children this process is somewhat delayed. After separation of the cord the skin of the umbilicus folds inward so as to protect and cover the umbilical wound until an epithelial coating makes it less susceptible to injury and infection.

### INFECTION OF THE NAVEL WOUND

Infection of the navel wound occurs during the first few days of life, either before or just following separation of the stump of the cord. The manner in which this infection may occur and its causes are discussed under Septic Infection in the New-Born. Omphalitis may be mild or severe. Redness, swelling, and infiltration of the umbilicus are here more or less marked, and a seropurulent or purulent discharge soon makes its appearance. The skin around the umbilicus may be excoriated and the subcutaneous tissues may become infected so that abscesses may form. As the inflammation subsides the umbilical pocket may be the site of an ulcer; in this condition the parts remain tender, somewhat swollen and continue to discharge pus.

**Umbilical Vegetations.**—Umbilical vegetations, or granuloma, are a not uncommon result of infection of the umbilicus. After the stump of the cord has fallen off a small red granulating mass is noticed, which gradually increases in size, protruding through the umbilical opening. This small

red tumor is associated with a serous discharge not infrequently tinged with blood. This tumor may become as large as the end of one's finger; it is commonly pedunculated and is bright or dark red in color.

**Gangrene of the Umbilicus.**—Gangrene of the umbilicus is rare and occurs most commonly in feeble, malnourished children. In this condition the umbilical wound assumes the appearance of ordinary gangrene. The gangrenous process spreads not only into the surrounding skin and subcutaneous tissues, but also involves the umbilical vessels and produces a fatal peritonitis or sepsis.

Infection of the navel wound resulting in inflammatory processes may, as previously noted, involve the umbilical vein and the umbilical artery, producing phlebitis and arteritis, and thereby readily leads to general septic infection of the new-born.

**Treatment of Infections.**—The prophylactic treatment is purely obstetrical and consists in the proper care of the cord. A few minutes following delivery, after normal respiratory efforts have been established, and the change in circulation has occurred, the cord is to be carefully ligated with tape and then cut. This should be done with clean hands and clean instruments. Following the bathing of the infant, the cord is to be dressed with proper surgical precautions, and thereafter redressed as often as may be necessary to keep the wound clean and prevent infection. If more than the normal amount of inflammatory reaction occurs in the umbilical wound, either before or after the separation of the stump, it is to be carefully cleansed with a 1 to 1,000 bichlorid solution or with a 1 to 4 peroxid of hydrogen solution and then covered with some antiseptic powder, which is to be held in place by a pad of gauze, covered with absorbent cotton and a retaining bandage. The importance of promptly treating all umbilical infections by the most approved surgical methods has been emphasized in the treatment of Septic Infections of the New-Born. Where abscesses occur they are to be opened and properly drained. Ulcers may be treated with weak astringent powders or they may be cleansed and treated with a ½ per cent. solution of nitrate of silver. Granuloma should be cauterized with the solid stick of nitrate of silver. If they do not yield to this treatment they should be curetted or cut away, and the wound thus produced covered with aristol or some other antiseptic powder, held in place by pieces of gauze and adhesive plaster.

## UMBILICAL HEMORRHAGE

A slight umbilical hemorrhage may occur from improper ligation of the cord, from its premature separation, or from injury to the umbilicus during the early days of life. Hemorrhages of this character are easily controlled and are of little pathological importance. The change in the circulation which occurs at birth so diminishes the blood pressure in the blood vessels of the cord that serious traumatic hemorrhage, occurring in an otherwise normal infant, is always a matter of gross negligence on the part of either the nurse or the physician. Persistent hemorrhage from the

umbilicus which fails to yield to simple treatment is therefore a matter of grave import and indicates serious constitutional disorder which has affected the capillary circulation of these parts in such a way that normal coagulation of the blood cannot be induced for the stopping of the hemorrhage. Hemorrhage of this character may, as previously noted, be a symptom of septic infection in the new-born or it may be one of the early manifestations of syphilis or hemophilia. It is sometimes associated with pronounced jaundice. It is always an indication of profound and dangerous constitutional disturbances and is frequently accompanied by other evidences of a general hemorrhagic diathesis, such as hemorrhages from other mucous membranes and petechial hemorrhages into subcutaneous tissue.

**Treatment.**—The simple forms are readily controlled by bandages making pressure over the part, which may be saturated with a 1 to 1,000 adrenalin solution. The grave forms of hemorrhage, however, are to be treated as recommended under Septic Infections in the New-Born. The syphilitic cases should receive anti-syphilitic treatment.

FIG. 16.—UMBILICAL HERNIA.

### UMBILICAL HERNIA

A slight dilatation of the umbilical ring, with protrusion of the intestine forming a small tumor the size of the end of one's little finger, is very common in premature and congenitally weak infants. It also occurs in older infants who are rachitic or exceedingly malnourished, and who have suffered from gastrointestinal disturbances, resulting in marked abdominal distention. Slight hernias, both umbilical and inguinal, are very commonly seen in pot-bellied, malnourished, rachitic infants. These protrusions are greatly aggravated by crying and coughing.

**Treatment.**—In the form of umbilical hernia which makes its appearance just after birth the prognosis is good and the treatment is simple.

The ordinary abdominal bandage of the infant holding a pad of gauze over the umbilicus may be applied more snugly than usual. This is all that is necessary during the first few weeks of life. Later the hernia should be reduced and held within the abdominal walls by a strip of adhesive plaster. This strip of plaster should extend across the abdomen and as it is applied the abdominal walls at the umbilicus should be folded in a vertical direction over the umbilicus, so that the approximated folds held by the adhesive plaster cover the umbilicus and prevent the hernial protrusion. The adhesive plaster should be renewed every three or four days, and if ulceration or irritation of the umbilicus or of the surrounding skin has occurred the treatment is to be discontinued until these parts have been entirely healed. The hernia in these cases may also be held in by making a round pad of thin wood about the size of a quarter, covering it with a piece of soft cloth and holding it in position over the umbilicus with adhesive plaster. With treatment of this kind applied over a period of five or six weeks, the umbilical opening commonly closes and a permanent cure results. In some instances, especially in malnourished infants, the hernia persists throughout infancy and perhaps gradually increases in size. A surgical operation is necessary for the cure of these cases.

There is a rare and much more serious form of *congenital hernia, due to arrested development.* In these cases the hernial sac at birth may be very large and filled with intestines and occasionally other abdominal organs, such as the liver, spleen, and kidney. These cases demand immediate surgical interference.

## MASTITIS

Enlargement of one or both mammary glands in the new-born is a not uncommon occurrence. It is most frequently seen during the second week of life. These swollen glands may secrete a milk-like substance, and on palpation they are found to be caked and more or less tender. In the majority of instances this tumefaction gradually disappears; the breasts become less tender, less swollen, and by the end of the third week of life have resumed their normal proportions. In other instances an infection of the gland occurs which causes it to become more inflamed, red, and swollen. This may produce a slight fever and after a number of days fluctuation may be discovered.

**Treatment.**—Previous to suppuration the swollen breasts are to be covered with gauze or lint, saturated with a weak solution of bichlorid of mercury, which is to be held in position by a carefully adjusted bandage. When fluctuation is discovered it is to be treated as any other abscess, by incision, proper drainage, and careful cleansing with antiseptic solutions.

## HOLT'S INANITION FEVER

Under the term "inanition fever" Holt has described a distinct clinical syndrome characterized by fever and nervous irritability. It occurs during the first five days of life.

**Etiology.**—The term "inanition fever," as Holt says, is not a satisfactory one. It is probably an autointoxication, due to the failure of the newly born infant to get sufficient fluid from the breast to flush out its kidneys and other excretory organs. It is clearly evident that the condition is commonly due to a deficiency in the breast milk. It disappears quickly when the milk secretion is established or when the infant is given water in sufficient quantities. Apart from determining the exact pathological cause of this syndrome there is little to add to the very clear description of its etiology, symptomatology, and treatment as given by Holt.

**Symptomatology.**—Holt says: "The symptoms are so uniform and so characteristic that they make for these cases of fever a class by themselves. The frequency with which this is seen is shown by the following statistics: Among two hundred infants taken successively at the Nursery and Child's Hospital twenty had fever during the first five days, reaching 101° F. or over, which was not explained by ordinary causes. In five hundred successive children born at the Sloane Maternity Hospital there were one hundred and thirty-five with a similar fever. It was seen in vigorous infants as well as in those who were delicate. The usual duration of the fever was three days, the temperature generally reaching the highest point upon the third or fourth day of life. In about two-thirds of the cases the temperature did not rise above 102° F.; in nine it was 104° F. or over, the highest recorded being 106° F. The fall was generally quite abrupt, although not always so. Daily weighings showed that the infant continued to lose weight while the fever continued and that the loss almost invariably exceeded by several ounces that of children who had no fever. The maximum loss noted was twenty-eight ounces. In quite a large number of cases it exceeded twenty ounces. As a rule, the infant began to gain in weight when the temperature remained at the normal point, but not until then.

"The symptoms presented by these infants were a hot, dry skin, marked restlessness, dry lips, and a disposition to suck vigorously anything within reach. With very high temperature there was considerable prostration and weakened pulse. In the less severe cases there were only crying and restlessness. The rapidity with which the symptoms disappeared when the children were wet-nursed or properly fed was very striking."

In addition to this symptom group I have commonly observed in these cases a marked diminution in the urinary secretion and occasionally anuria over a period of twenty-four or thirty-six hours, to be followed by the discharge of a small quantity of highly colored urine, occasionally tinged with blood.

**Diagnosis.**—This condition can scarcely be mistaken for anything except sepsis in the new-born. Holt's fever occurs during the first five days of life; sepsis occurs most commonly during the second week and occasionally later. The promptness with which Holt's fever responds to proper treatment and the seriousness of the septic syndrome with continuance of the fever, prostration, and other associated symptoms make the diagnosis clear.

**Prognosis.**—This is good. All of these cases make a satisfactory recovery as soon as the proper treatment is instituted.

**Prophylaxis.**—In view of the prevalence of this condition, all infants during the first few days of life should be given small quantities of water at frequent intervals and special attention should be given to the condition of the mother's breasts, to ascertain whether the milk secretion is being established at the normal time. In cases where there is a delayed establishment of the milk secretion weak solutions of skimmed milk, 1 to 4, or breast milk should be given until the milk secretion of the mother has been fully established.

**Treatment.**—The curative treatment of this condition, as Holt has said, is to give the infant water at short intervals and to supply it with food in the form of breast milk from a wet-nurse or with weak mixtures of cow's milk. In the event that the secretion of milk in the mother's breast is not properly established, the infant should be fed permanently upon the breast milk of a properly selected wet-nurse.

## CHAPTER VIII

### DISEASES OF THE NEW-BORN (*Continued*)

### TETANUS NEONATORUM

Tetanus of the new-born, like tetanus in the adult, is an acute infection produced by the tetanus bacillus.

**Etiology.**—The tetanus bacillus finds its portal of entrance at the umbilical wound, and in this pocket it multiplies rapidly and forms its specific toxin (tetanotoxin), which is rapidly disseminated throughout the body. The tetanus toxin has a special predilection for nerve tissue and probably unites in organic combination with the material forming the motor cells of the spinal cord and medulla. This produces an intense reflex excitability and irritability of the motor cells of the spinal cord and of the medulla oblongata. The irritability of these tissues becomes so great that the slightest reflex cause will excite violent tonic muscular contractions. The tetanus bacillus is believed to confine itself almost entirely to the umbilical pocket and there produce the toxin, the absorption of which is responsible for the profound toxic symptoms of this disease. The fact, however, that the blood of patients suffering from tetanus is capable of transmitting the disease when injected into animals indicates that along with the toxin thus injected there must be at least a few tetanus bacilli.

The tetanus bacillus is found in the superficial layers of the earth and is much more prevalent in some localities than in others.

Tetanus is most common among the class of people who live in uncleanly surroundings. It is a dirt or filth-borne disease and is carried to the umbilicus by dirty hands, dirty clothing, or by anything that may carry

dust or other dirt to the wound. With individuals living amidst dirty surroundings it is possible that the tetanus bacillus may be carried through the air on particles of dust to the umbilical wound, which, especially after the stump of the cord has been separated, furnishes so suitable a soil for its growth. In rare instances the tetanus bacillus may find an entrance through wounds or raw surfaces other than that of the umbilicus, but that this is a very unusual occurrence is indicated by the fact that tetanus in the young infant is confined almost exclusively to the first three weeks of life, when the umbilical wound is still open. It may occur during the first days of life, but is much more common during the second week, when the cord has separated and the umbilical wound is open. It becomes less frequent during the third week of life as all irritation about the umbilicus gradually disappears, and after the third week, with the umbilical wound entirely healed, it is very rare.

**Symptomatology.**—Nervousness, irritability, and sleeplessness are the usual premonitory symptoms. These are followed by difficulty in nursing; the child lets go of the breast with a sudden cry after a few attempts at sucking. The lower jaw gradually loses its motility and in putting the child to the breast it is noticed that there is a firm and tonic contraction of the muscles which causes it to become locked in such close proximity to the upper jaw that food, water, and medicines can be introduced into the mouth only by means of a medicine dropper. This condition of trismus is an early and characteristic symptom in practically every case of tetanus neonatorum. In the beginning these muscular spasms are followed by periods of relaxation, but, on attempting to feed the infant by putting the nipple or a spoon between its gums, the lower jaw is again thrown into a condition of spasm. These attacks of trismus continue to recur with greater frequency and are more prolonged as the disease advances, so that within a short period of time, usually a few days, the lower jaw is continuously locked in close proximity to the upper jaw by the tonic muscular contractions.

Spasms of the muscles of the face, which soon become associated with the trismus, produce a very characteristic expression. The forehead is wrinkled, the eyes closed, and the mouth puckered. Gradually the muscles of the neck, back, abdomen, and extremities are affected, and the tetanic contraction of these muscles produces retraction of the neck, opisthotonos, and stiffness of the entire body. All the joints of the arms and legs are in a condition of flexion. The muscular rigidity reaches its maximum in from one to three days. In severe cases the child may die before the lower extremities are involved or in mild cases recovery may occur without the disease extending to the arms and legs. Swallowing becomes more difficult and finally impossible; respiration is embarrassed as a result of spasm of the diaphragm. Throughout the disease the tetanic contractions are greatly exaggerated by slight reflex causes, such as attempting to feed the child or handling it for absolutely necessary purposes.

The temperature is of no diagnostic value. At the onset and just before

death it may be as high as 105° or 106° F. During the course of the disease it may be subnormal. The child lies in the rigid condition above described, making no outcry because of the spasm of the laryngeal muscles; its breathing becomes more and more superficial and irregular; its pulse more feeble and rapid; the muscles of its body become more continuously and more rigidly contracted, until death occurs from asphyxia or exhaustion. In those cases which are fortunate enough to recover there is a gradual recession of the symptoms, the period of relaxation between the spasms becomes greater and the trismus is less marked, and the child less readily responds to reflex excitation.

**Diagnosis.**—There should be no difficulty in the diagnosis of tetanus in infancy. The only conditions for which it may be mistaken are meningitis and brain injuries producing spastic paralysis, and sometimes opisthotonos, but in these cases the characteristic symptom of trismus is absent and the tonic muscular contractions do not recur in spasms excited by slight reflex stimuli.

**Prognosis.**—Nearly all of these cases die. By some writers the percentage of recovery is placed as high as 30 per cent. and others report only 2 or 3 per cent. of recoveries.

**Treatment.**—The prophylactic treatment pertains to the care and management of the stump of the cord and of the navel wound which results from its sloughing off. This treatment is of special importance in infants who are born under dirty surroundings; that is to say, under conditions where there is a probability or possibility that the navel wound may be inoculated with filth, dust, or dirt containing the tetanus bacillus. The prophylactic treatment, therefore, is purely obstetrical, and all of the conditions necessary to absolute asepsis should be rigidly enforced in cutting the cord, ligating the stump, and dressing the wound. All of this must be done with clean hands, clean instruments, and afterward the navel is to be so dressed with a dry antiseptic dressing that it is impossible for it to be contaminated by dirty surroundings. It is especially important to remember that the navel wound is to be dressed and redressed for three weeks, or until it has entirely healed. It is during the second week after the cord has sloughed off that the navel is to be especially protected from contamination with anything that may act as a carrier of the tetanus bacillus. If infection of the umbilical wound occurs it is to be carefully washed out with a 1 to 4 solution of peroxid of hydrogen or a 1 to 1,000 solution of bichlorid of mercury, then carefully dried, and dressed with some antiseptic powder.

In the treatment of the disease itself it is wise to use tetanus antitoxin with the onset of the first symptoms. This antitoxin can do no harm even in large doses. It acts by combining with the toxins of this disease and thereby neutralizing their poisonous effect upon the nerve centers. To be of benefit, therefore, it must be given early and in large doses. It is commonly introduced by lumbar puncture into the spinal canal or into the subcutaneous tissues in the same way as diphtheria anti-

toxin. From 5 to 10 c. c. may be introduced into the spinal canal, following a lumbar puncture which has drained away that amount of fluid. The technique of this operation is exactly the same as that used for introducing anti-meningitis serum in cerebrospinal meningitis. At the same time 10 or more c. c. of antitoxin may be introduced subcutaneously. The subcutaneous injection may be repeated at intervals of six hours and the spinal canal injection at intervals of twenty-four hours for two or three days. If no appreciable result has then been produced by the treatment it should be discontinued.

The symptomatic treatment consists in keeping the child as quiet as possible, touching it only when necessary, and shielding it from all noises. Its nourishment should be breast milk. When it can no longer nurse the breast it should be fed breast milk with a medicine dropper, or the same food may be introduced into the stomach through a catheter which is passed through the nose and down the esophagus. When these children, however, have reached a stage when deglutition is impossible, little is to be accomplished from introducing food in this way.

Chloral is by far the most valuable drug for controlling the muscular contractions and making the patient more comfortable. It is to be given in 1 or 2-grain doses every two or three hours, as indicated, to relieve the symptoms. When the infant can no longer swallow, chloral, in 2 to 4-grain doses, should be given by the rectum. In milder cases bromid of potash may be used with or instead of the chloral.

## ICTERUS NEONATORUM

This is the form of jaundice so common in the new-born. It runs a benign course and is of importance from the standpoint of differential diagnosis. It must be differentiated from the grave forms of jaundice due to occlusion of the bile ducts and to the jaundice which occurs as a symptom of sepsis and cirrhosis of the liver.

**Etiology.**—The etiology of icterus neonatorum remains obscure, notwithstanding the many ingenious theories which have been offered in its explanation. It has been suggested that the excessive destruction of red blood corpuscles during the first days of extrauterine life and the abundant blood supply to the liver at this time cause the liver to form an excess of bile, part of which is reabsorbed, passes into the blood stream and produces jaundice. A part, however, of this excess of bile passes through the bile ducts into the intestine, and for this reason the intestinal symptoms, which are such an important part of the symptom complex in obstructive jaundice, are almost or entirely absent in this condition. This form of jaundice can scarcely be spoken of as a pathological condition, since it is the result of physiological causes acting under new and perhaps exaggerated conditions. It occurs to a greater or less degree in from 60 to 80 per cent. of all newly born infants, and some observers place this percentage even higher. It is more common, or at least more severe and prolonged, in feeble, malnour-

ished infants, and especially in those born prematurely. For this reason it is more commonly seen in public maternity hospitals and foundling institutions than in private practice.

**Symptomatology.**—The jaundice may appear a few hours after birth, but is commonly not recognized until the second or third day. It remains at its height but a few days and then begins to slowly disappear, so that in the great majority of cases no trace of it is left after the eighth or ninth day. In some instances, however, the jaundice may persist for several weeks. This is much more likely to occur in premature and malnourished infants. The sallowness makes its appearance first on the face, chest, and back, and in the more marked cases the yellow color deepens and the jaundice extends to other parts of the body. The constipated, putrid, clay-colored stools, so characteristic of obstructed jaundice, are nearly always absent. The discharges from the intestinal canal are almost normal, or perhaps modified in the more marked cases by slight intestinal indigestion.

The conjunctiva is slightly tinged with yellow, but not so markedly as in obstructive jaundice. The diagnosis in this form of jaundice is made rather by the sallowness of the skin than by the yellowness of the conjunctiva. When the jaundice is at its height, however, in the marked cases, the conjunctiva has a distinctly yellow color. The urine in such cases is dark in color and produces a dark yellow stain on the napkins, and bile can sometimes be demonstrated in it by the ordinary chemical tests. The discoloration of the urine, however, and its reaction to the ordinary tests for bile is never so marked in this condition as in obstructive jaundice, and early and late the urine may furnish no evidences of containing bile.

Infants with icterus neonatorum present no constitutional symptoms of illness. They are, as a rule, happy, take their food in a normal manner, and, apart from the evidences of jaundice above given, are apparently well. The fact that premature infants and infants suffering from more or less profound malnutritions have a more marked and more prolonged icterus is an evidence that these conditions exaggerate the jaundice, rather than that the jaundice aggravates the malnutritions.

**Treatment.**—This condition runs a benign course, terminates in recovery, and is perhaps not influenced by therapeutic measures. It is wise, however, in these cases to clear out the intestinal canal with a little chalk mercury, followed perhaps by milk of magnesia.

### OCCLUSION OF THE BILE DUCTS

Occlusion of the bile ducts in the new-born is rare; it may be due to catarrh of the mucous membrane or congenital malformations. The most common malformation is obliteration of the common gall duct; in some instances this duct may not be entirely absent, but almost occluded. The cystic duct and gall bladder may be rudimentary or absent.

**Symptomatology.**—The symptoms are those of obstructive jaundice. The sallowness of the skin becomes a deeper and more pronounced yellow and the whole body has a markedly jaundiced hue. In rare cases where the

obstruction is not complete the jaundice may not be so pronounced and may vary in degree from time to time. These are the cases which may live for months, slowly dying of malnutrition. In the great majority of instances, however, the pronounced yellowness of the skin is associated with a well-marked yellowness of the conjunctiva; the urine contains bile, which may readily be demonstrated by ordinary chemical reactions, it is dark in color and stains the napkin a yellowish brown. The discharges from the intestine gradually become clay colored, have an offensive odor, and are, as a rule, dry and constipated. The liver is enlarged and not infrequently the spleen may be easily palpated. The child loses in weight and strength and presents every appearance of being extremely ill. Indigestion and intestinal toxemia with an associated elevation of temperature are commonly present. As the malnutrition progresses the child becomes listless and lethargic and not infrequently develops a hemorrhagic diathesis. Bleeding may occur from mucous membranes and purpuric spots may appear over the body. These infants commonly die from malnutrition or autointoxication within a few weeks; in the less severe cases death may be postponed for some months. The rare cases of catarrhal jaundice may be prolonged for weeks and ultimately recover.

### OTHER FORMS OF ICTERUS OCCURRING IN THE NEW-BORN

Jaundice is a symptom of septicopyemia occurring in the new-born. The jaundice due to this cause is considered under Septic Infection.

Jaundice may also occur as a symptom of congenital syphilis in the new-born. This is a comparatively rare cause at this period of life. The jaundice in this condition is due to cirrhosis of the liver. The interstitial hepatitis compresses the bile ducts and interferes with the outflow of bile. The symptoms in this form of jaundice resemble those of the milder forms of obstructive jaundice produced by congenital occlusion of the bile ducts. The skin, conjunctiva, and urine show the ordinary signs of jaundice and a hemorrhagic tendency may develop late in the disease; the clinical picture produced does not in the least resemble icterus neonatorum. It is to be differentiated from congenital obliteration of the bile ducts by its slower onset, less severe and more prolonged course, but more especially by the existence of other evidences of congenital syphilis.

The prognosis of syphilitic jaundice is bad. Antisyphilitic treatment may prolong but it cannot save the lives of these infants.

## OPHTHALMIA NEONATORUM

Ophthalmia neonatorum is an inflammation of the conjunctiva occurring in the new-born.

**Etiology.**—The gonococcus is the cause of this disease in perhaps 70 to 80 per cent. of all cases. Infection of the conjunctiva with other pus-forming organisms, such as streptococci, staphylococci, and pneumococci, is responsible for the remaining cases. Infection results from the direct

inoculation of the conjunctiva with one or the other of these pus-forming organisms and usually occurs during the birth of the child.

Gonococcus and other forms of vaginitis and urethritis in the mother may produce this disease in the infant. Occasionally infection may be carried to the conjunctiva of the infant by the hands of the obstetrician or nurse, either during or after labor.

Postpartum infection is comparatively rare and is due to gross carelessness or negligence on the part of those who have the care of the infant. This is much more likely to occur in hospitals and other institutions than in private families, but the transference of septic infection from other patients to the eyes of healthy infants is now fortunately rare, even in public lying-in institutions and foundling asylums.

**Symptomatology.**—Since infection nearly always occurs during birth the symptoms commonly make their appearance before the fourth day. If conjunctivitis develops after the fifth day it is almost certainly due to postnatal inoculation. The disease announces itself with redness and swelling of the conjunctiva of one or both eyes. Very commonly the eyelids become so swollen and edematous that the eyes are closed and the infant no longer has the power of opening them. Through the palpebral fissure there issues a thin, yellowish discharge. When the lids are pressed apart by the fingers both the ocular and palpebral conjunctiva are seen to be violently inflamed, much swollen, gathered in folds, and covered with a purulent mucus. The folding of the conjunctiva is especially noticeable, where it crowds over the corneal margin. As the disease progresses the eye becomes more swollen and the discharge changes to a yellow, creamy pus which exudes in great profusion as the palpebral fissure is opened. At this stage of the disease there is great danger that ulceration of the cornea may occur. These ulcers may be central or marginal, the latter may be hidden beneath the folds of the overhanging conjunctiva. The appearance of corneal ulcers adds great gravity to the case. These ulcers may perforate, and prolapse of the iris, loss of the aqueous humor, and panophthalmitis with permanent loss of vision may result. This disease is, in fact, responsible for about 30 per cent. of the cases of blindness found in public institutions. In those cases that recover under suitable treatment, without corneal involvement, the first favorable indications are gradual decrease in the swelling and thickening of the lids. The palpebral fissure is more readily opened and the eye is more easily irrigated, and gradually the redness and swelling of the conjunctiva disappears. In these favorable cases convalescence is established within two or three weeks.

In the gonococcus cases the inflammation is much more violent, the dangers of corneal ulceration greater, and the disease runs a more prolonged course than in the simple cases produced by other pus-forming organisms.

**Diagnosis.**—The differential diagnosis of gonorrheal from other forms of ophthalmia is made by the history of the case with reference to possible gonorrheal infection and by the violence of the inflammation. In doubt-

ful cases a bacteriological examination may determine the character of the infection.

**Prognosis.**—Cases that are seen early and subjected to proper treatment, as a rule, terminate in complete recovery. Neglected cases, especially of the gonococcus type, commonly result in corneal ulceration with permanent loss of vision.

**Prophylaxis.**—The prophylactic treatment is especially important in institutional practice, where gonorrheal and other forms of vaginitis are so commonly seen in the mother. In institutions, therefore, it is perhaps wise to employ in all cases the preventive treatment recommended by Credé. Directly after birth the child is carefully washed, and during this process special care should be taken to avoid contaminating the conjunctiva with the bath water or with wash-rags used on other parts of the infant's body. Following this, one drop of a 1 to 2 per cent. solution of nitrate of silver is carefully dropped into each eye; experience favors the weaker solution. In private practice the silver solution is not, as a rule, indicated unless the mother has a vaginal discharge. In all cases, whether in institutional or in private practice, where the mother has a purulent or other vaginal discharge the vagina, for days prior to the delivery of the child, should be carefully douched with alkaline antiseptics.

In private practice where the vaginal conditions in the mother are normal no prophylactic treatment is necessary except the careful washing of the infant's eyes with a weak boric acid solution immediately after delivery. Where one eye only is infected it is of the greatest importance to protect the other eye from inoculation. This perhaps can best be done by covering the well eye with cotton and lint and carefully bandaging it so as to prevent its accidental inoculation with the pus from the infected eye. Such a bandage should be removed and reapplied daily, to see that the eye has not become infected.

**Treatment.**—The treatment of ophthalmia neonatorum is of such great importance that to carry it out properly requires the constant attention, day and night, of capable nurses, and where the inflammation begins violently and the indications are that the disease is of gonococcic origin it is best to have the advice of an oculist.

The treatment consists in the constant application of ice-cold compresses. These are made of pieces of lint or gauze large enough to cover the eye, which are kept on a piece of ice floating in a saturated solution of boric acid or a 1 to 5,000 solution of bichlorid of mercury. These pieces of cloth are transferred from the ice to the inflamed eye every few minutes and changed from time to time as cleanliness demands. During this treatment, at intervals of one or two hours, the palpebral fissure should be opened and the pus thoroughly washed out of the eye by douching it with a boracic acid solution, and once in twenty-four hours a 2 per cent. solution of silver nitrate should be instilled into the eye. Within a few days the inflammation should begin to subside, and with this improvement the cold applications may be applied interruptedly instead of continuously, but the

irrigations with boric acid solution and the instillations of silver nitrate should be continued.

If the cornea is involved the case should be referred to an oculist. In these cases a 1 per cent. solution of atropin should be dropped into the eye from time to time until the iris is widely dilated, and throughout the treatment this dilatation is to be maintained. In these corneal cases the irrigation of the eye with mild antiseptic washes and the use of silver nitrate solutions as above noted are to be continued, but the cold applications are of doubtful efficacy.

## CHAPTER IX

### BIRTH INJURIES

### CEPHALHEMATOMA

**Symptomatology.**—This condition is due to an injury of the subperiosteal blood vessels occurring during birth. The tearing of these blood vessels results in the pouring out of blood between the bones of the skull and the periosteum. This produces a swelling of the scalp which commonly appears between the first and the fourth day of life. It is usually located over one and rarely over both parietal bones. It may be large enough to cover the whole, but in most instances only a part, of the parietal bone, and it is limited by the parietal sutures. It may reach the size of a large orange. The overlying skin undergoes no change nor is there any tenderness or other evidence of inflammatory action. The tumor is soft and fluctuating, so there is never any doubt of its fluid contents. After a time, at the circumference of the tumor, a hard, distinct elevation forms. The tumor gradually increases in size for a period of perhaps one week; thereafter it may remain stationary for a few days and then very gradually diminish in size. It commonly requires from two to four months for its complete disappearance. As it commences to diminish in size it loses its tenseness and becomes soft and flabby.

**Diagnosis.**—The differential diagnosis from caput succedaneum should cause no embarrassment. In this latter condition the swelling of the scalp is not only present but is at its height at birth. It does not fluctuate, is soft and flabby at all times, and begins to disappear on the second or third day. The differential diagnosis from hernia of the brain or its membranes presents no difficulties, as these symptom groups are quite distinct. From abscess of the scalp it may be differentiated by the absence of inflammation. If the tumor is red and tender and accompanied by constitutional symptoms the introduction under aseptic precautions of an aspirating needle will determine the character of the contained fluid. Rarely cephalhematoma may be associated with a hemorrhagic diathesis resulting from some severe constitutional disorder. These are the only cases, and they are very rare, in which the prognosis may be unfavorable.

**Treatment.**—As a rule no treatment is necessary; spontaneous recovery occurs. If there be delay in the disappearance of the tumor it may be wise to inquire whether or not the cephalhematoma has been converted by infection into an abscess. This may be determined by the introduction of an aspirating needle. In the event that pus is found the abscess is to be evacuated by free incision, thoroughly drained, packed with gauze, and thereafter treated as any other abscess.

## HEMATOMA OF THE STERNOCLEIDOMASTOID MUSCLE

This condition results from the stretching and tearing of this muscle in such a way as to produce a hemorrhage into its sheath. This injury is comparatively rare, but occurs most commonly in breech presentations.

**Symptomatology.**—Soon after birth it is noted that the child has a stiff neck. Its head is turned to the affected side. This torticollis results from contraction of the injured sternomastoid muscle, and within or along this tense muscle a small tumor may be felt. The part is painful to touch and the child cries when an attempt is made to overcome the deformity. After a time the hematoma is absorbed, but the contraction of the muscle may remain for many months and in some cases it is permanent.

**Treatment.**—After some months, when all soreness and tenderness have disappeared, an attempt should be made to overcome the deformity by massage and passive movements. If these measures fail the patient should be referred to an orthopedic surgeon, that the deformity may be overcome by operative measures.

## BIRTH PALSIES

Birth palsies may be central or peripheral in their origin. Central palsies are described under Cerebral Palsies. Peripheral or obstetrical palsies occur as two distinct clinical types, known as facial paralysis and upper arm paralysis.

### FACIAL PARALYSIS

This is a paralysis of the seventh or facial nerve produced by injury during birth. It is commonly due to the pressure of the blades of the forceps. In most instances it is unilateral and the diagnosis is made by a lack of symmetry in the two sides of the face, due to paralysis of the muscles of one side. This is much more noticeable when the face muscles are in action. This condition is of little pathological importance, as the paralysis disappears spontaneously in two or three weeks. Very rarely the injury to the nerve may be so great that a permanent paralysis results.

Treatment as a rule is unnecessary. In those cases, however, in which the paralysis persists the muscles should be exercised and atrophy delayed or prevented by the systematic use of massage and electricity as outlined under Cerebral Palsies.

## UPPER ARM PARALYSIS

*(Duchenne-Erb's Palsy)*

**Etiology.**—This is due to some injury of the fifth, sixth, and seventh cervical nerves during birth. It is more common after breech presentations. It may be produced by pulling or twisting the arm or shoulder or by axillary pressure from a blunt hook or the obstetrician's finger. Whatever may be the modus operandi of the development of this paralysis, the fifth and sixth cervical nerves are so pressed upon, stretched, or twisted as to put them out of function, and a motor paralysis of the muscles which they supply results. The deltoid, biceps, brachialis anticus, and supinator longus muscles are most commonly involved.

**Symptomatology.**—As a rule only one arm is affected. It is noticed within two or three days after birth that this arm hangs limp and motionless and is rotated inward. The paralysis in these cases is of the upper arm type described by Duchenne and Erb. The muscles of the wrist and hand are not affected. The paralysis is almost exclusively motor; there is little or no disturbance of sensation. In the great majority of cases recovery slowly takes place within two or three months. In a few cases the paralysis persists, the affected shoulder droops, muscular atrophy slowly takes place, and after a time the shoulder and upper arm are markedly lacking in development. With the wasting of the upper arm muscles there is also more or less lack of development of the bones, so that the arm is not only shriveled, but shorter than its fellow of the opposite side. Contractures of the muscles of the lower arm and hand develop. In rare instances subluxation of the head of the humerus takes place and greatly increases the deformity. In the worst cases the reaction of degeneration is finally followed by a failure to respond to either the galvanic or faradic current and the arm remains comparatively useless throughout life.

**Treatment.**—Within two or three weeks after birth gentle but systematic massage should be begun. This is to be applied especially to the muscles of the shoulder and upper arm. If at the end of the third month convalescence has not been established, the faradic current should be used in connection with massage to exercise the paralyzed muscles, and in the event that the muscles do not respond readily or normally to the faradic current the galvanic current should be substituted. This treatment should be persisted in for months, and in the event contractures occur the advice of an orthopedic surgeon should be sought. Many of these cases are greatly benefited by surgical operations and by orthopedic appliances to overcome contractures and develop weak and degenerating muscles.

# SECTION III

## *INFANT FEEDING*

## CHAPTER X

### MILK IN ITS RELATION TO INFANTILE NUTRITION

Human breast milk is the ideal infant food, evolved by natural laws to suit the immature digestive organs of the human infant and to furnish the exact nutritional elements necessary for the rapid growth and development of the human organism. Its various ingredients, in their digestibility, their chemical composition, their total quantity, and in their relative proportion to other ingredients, are just what they should be; and if it were possible to feed every human infant upon normal human milk for the first nine months of its life the whole problem of infant feeding, which is the most important single subject claiming the attention of pediatricians today, would dwindle into comparative insignificance.

Cow's milk is also an ideal food for the young of its kind, and its various ingredients, including their chemical composition, their digestibility, their quantity, and their relative proportion to other ingredients, are suited to the purposes cow's milk is intended to serve; namely, to furnish nutrition to the young calf and to develop its digestive organs so as to prepare them for the food which is to follow. Thus it is plainly evident that the breast milk of individual mammals is suited to the development of the young of its kind, but is not necessarily suited and has not been evolved by nature to nourish and develop the digestive organs of a different species.

Milk is composed of fat, protein, carbohydrates, mineral salts, water, ferments, alexins, antitoxins, etc. It is a live fluid with definite chemical and biological properties which can only be understood by a careful chemical and physiological study of its various ingredients in relation to infantile nutrition.

**Fat.**—The fats which compose about 4 per cent. of both human and cow's milk are found in fat molecules suspended in the form of an emulsion. Their composition is very complicated. They contain neutral fats and fatty acids. The larger molecules contain a greater percentage of volatile fatty acids and the smaller ones more oleic acids, so that in

skimming milk, the larger fat globules rising to the surface above the smaller ones, a greater percentage of volatile fatty acids is skimmed off and more oleic acid left in the small globules. These fatty acids are mixed with glycerin and therefore occur in the form of glycerides. Butyric, palmitic, and stearic acids are the most important of the fatty acids. A small portion of the fatty acids are compounded with albumin in the form of lecithin, which may exist outside of the fat molecule. Cow's milk contains a greater percentage of volatile fatty acids and a less percentage of oleic acid than human milk, and the fat in the former is in coarser emulsion and separates more easily than in human milk. This difference in the composition of the fats of the two milks may in part explain the fact that the human infant may digest and assimilate 4 per cent. of fat in woman's milk and yet fail to digest 2½ per cent. of fat in cow's milk, and it may also explain why cow's milk, with its excess of volatile fatty acids, may predispose to acid intoxications in infancy, since these acids may be readily converted by hydrolysis into diacetic acid and acetone.

In the natural food of the human infant fat is a most important agent in keeping up the heat and furnishing the energy for cellular work. It is the fuel of the cells and is furnished in large quantities because of the unusual cellular activity which occurs at this time of life. It is the most important factor in increasing the weight of the body during early infancy and is stored in large amounts in the subcutaneous tissues to serve emergency purposes. These storehouses are drawn upon when there is a failure in fat digestion or fat assimilation. The nervous system which is so immature at birth and which develops so rapidly during the first year of life demands a large amount of fat for its proper development. The fat forms an important element of nerve structures, and there is no part of the infantile anatomy which suffers more seriously or more profoundly than does the nervous system when the digestion and assimilation of fats is interfered with. The bony structures also depend in part for their development upon a proper amount of fat in the tissues. The dangers from *too little fat* in the food are therefore most apparent, resulting in lack of development, especially in nervous and bony structures, loss of weight, anemia, and malnutrition. One should also remember that *an excess of fat* in the food of the infant may produce constipation or diarrhea with fat-stools and more or less serious gastrointestinal and nutritional disturbances; the serious "food injuries" which result from an excess of fat are very uncommon except in those cases where both the fats and the sugars are given in excess at the same time. When the sugar percentage of a food is high an excess of fats is likely to cause more or less serious digestive disturbance and *vice versa.* It is, therefore, sometimes difficult to tell whether the child has been made ill by the fats or the sugars, since, as a rule, improvement follows the elimination of either the fats or sugars from the diet.

**Protein.**—The chief proteins of milk are casein and lactalbumin; lactoglobulin, lactoprotein, and nuclein occur in smaller quantities. Under the term whey proteins all the proteins of milk except casein are grouped. In

woman's milk the whey proteins predominate over the casein in the proportion of 2 to 1, but in cow's milk the proportion is as 1 to 6. Chemistry has not as yet made any practical or important differentiation between the whey proteins of woman's and cow's milk, but we have in the quantities of casein and soluble albumins they contain two entirely different milks. The most important difference lies perhaps in the chemistry of the two caseins. This difference is recognized by the manner in which they react to the same ferments and reagents. In the stomach of the human infant the calcium casein of cow's milk (the form in which casein exists in cow's milk) is readily precipitated by rennet, in the presence of a slight amount of acid, into a clot of calcium paracasein, and later, as the hydrochloric acid is secreted in larger quantities, into hydrochlorate of paracasein and calcium; the calcium being separated from the paracasein clot by the hydrochloric acid. This clot is larger and tougher than the clots which occur in the infant stomach from the action of the same reagents on human milk. In human milk the paracasein clots and the hydrochlorate of paracasein clots are soft and light as compared with those of cow's milk.

Casein is rarely the cause of intestinal disturbance. On the other hand, Finkelstein and Meyer have apparently demonstrated that intestinal indigestion may be improved or controlled by increasing the quantity of the casein and diminishing the quantity of the sugar or fat in the milk. The digestibility of the casein of cow's milk depends largely upon the presence or absence of the conditions in the infantile stomach which cause its precipitation in small or large curds. *It is very easily digested and assimilated if large clot formations can be prevented.* If an alkali such as lime water or sodium bicarbonate, or an acid such as hydrochloric or lactic, be added to cow's milk before it enters the stomach, the alkali on the one hand or the acid on the other, by combining with the casein, may interfere with the action of the rennet in the formation of large clots, since rennet can only act in a slightly acid medium. The boiling of milk will also prevent the formation of large casein clots. In some instances it may also be necessary to reduce temporarily the amount of fat in the milk, so as to prevent its entanglement in the meshes of the clot. The danger, therefore, from an excess of protein lies in the fact that we are not always able to control the factors which cause clot formation, and for this reason it is sometimes necessary to diminish the amount of protein to a point where clot formation will not interfere with the intestinal digestion of the casein. In such instances it may be necessary to substitute the whey proteins in part for the casein so that the protein content of the food may not fall below the absolute nutritional demands of the body. In doing this, however, it should be remembered that in infants suffering from digestive disturbances the whey of cow's milk may aggravate the trouble. The human infant may digest the various food elements of cow's milk when they are held in the whey of human milk and may fail to digest them in the whey of cow's milk.

The casein of cow's milk contains 53 per cent. of carbon, 15.65 per cent.

of nitrogen, 7.06 per cent. of hydrogen and 0.85 per cent. of phosphorus, and 0.78 per cent. of sulphur. The general composition of proteins with the large per cent. of nitrogen they contain makes them absolutely necessary for cell growth and cell life. They furnish the material in large part from which the cells of the body are built up, and with the continuous cellular activity and nitrogenous waste there is a demand in the rapidly growing body of the infant for sufficient protein in the food not only to supply the cell waste, but to furnish material for the growth of new cells. The fats and carbohydrates, furnishing, as they do, the fuel from which the cells manufacture the heat and energy of the body, are necessary to prevent excessive nitrogenous waste, since the proteins are burnt up by the cells when the fats and carbohydrates are not present in sufficient quantity to supply them with fuel. This protection of the proteins by the fats and carbohydrates enables the cells to get on with the minimum amount of protein, a quantity sufficient to supply the normal nitrogenous waste and the material for new cells. The great loss that is sustained in protein food in the absence of fats and carbohydrates is indicated by the fact that twenty-two parts of protein are equal in fuel value to only ten parts of fat. It is very evident, therefore, that an artificial food should be carefully adjusted in its various ingredients to furnish the cells with sufficient energy and heat-producing food so that the proteins may not be wasted in serving this purpose. If the food of the infant should contain too little protein or if the fats and carbohydrates are diminished to such a low percentage that a portion of the moderate amount of protein taken be used for fuel, in both instances we would have a protein starvation, resulting in anemia, malnutrition, and general enfeeblement of cellular activity in all parts of the body.

**Carbohydrates.**—Human milk contains nearly 7 per cent. and cow's milk about 4 per cent. of milk sugar. This is the only carbohydrate that has been found in milk. There is little variation from day to day in the quantity of sugar in either woman's or cow's milk, this ingredient being subject to much less variation in quantity than the fats and proteins. Chemistry has not demonstrated any important difference in the composition or reaction to digestive ferments of the sugar found in the milk of different mammals. The milk sugar in human milk is especially adapted to supply carbohydrate food to the young infant. It does not readily ferment and is quickly converted into dextrose in the intestines. On the other hand, the milk sugar of cow's milk is, according to the German school, the most common cause of intestinal fermentation. It appears that milk sugar, when held in the whey of cow's milk, is less rapidly absorbed and more subject to fermentation than is maltose or dextrin. The great majority of normal infants, however, can readily digest it.

The carbohydrates next to the albumins are the most important food of the infant. They, like the fats, serve as a fuel for the cells making heat, and, what is more important, furnish the food which supplies energy to the cells. In their heat-forming capacity they are second to the fats

and in their energy-furnishing power they take the lead. It is an important physiological fact that the oxygen contained in carbohydrates is not only sufficient to oxidize their own hydrogen but to materially aid in oxidizing the waste products of the fat and protein molecules as they are broken down in the body, thus preventing an autointoxication. This is but another example of the interdependence of the protein, fat and carbohydrate molecules in serving the nutritional demands of the body, and makes plain the fact that we are acting wisely when we imitate nature in making an infant food by combining these food elements in proper proportions.

An excess of carbohydrates with other food elements (especially the fats) in normal proportions may result in diarrhea, loss of weight, fever, intestinal irritation, and catarrh. An excess of carbohydrates with the other food elements below normal may result in anemia, rickets, and general malnutrition. A deficiency in carbohydrates with other food elements in excess will, as a rule, overtax the digestive capacity of the infant and thereby produce digestive disturbances.

**Inorganic Constituents of Milk.**—Calcium, sodium, potassium, magnesium, phosphorus and iron are the most important inorganic constituents of milk. All of these, excepting iron, are present in both human and cow's milk in sufficient quantities to meet the nutritional demands of the growing infant.

Iron, as a necessary constituent of hemoglobin, is all-important in the oxidation processes which underlie body metabolism. The deficiency of iron in milk, which gradually increases as lactation proceeds, is made up during the first year of life from the stores of this mineral found in the liver and other organs of the newly born infant. At birth there is three times as much iron in proportion to body weight as in the adult. The partial iron starvation which occurs on a milk diet is not therefore of material consequence during the first year of life. But as the storehouses of iron become exhausted it is necessary to supplement the milk diet by such iron containing foods as eggs, fruit, and purees of vegetables, otherwise anemia and serious malnutrition may result.

The other inorganic constituents of milk play a no less important rôle in the body metabolism of the human infant than does iron. All of these are found in such organic combinations in human milk that they are readily assimilated in sufficient quantities to meet nutritional demands. It is also true that the normal human infant can, as a rule, assimilate sufficient quantities of the inorganic constituents of cow's milk. The fact that a smaller percentage of the salts of cow's milk is assimilated is offset by the fact that they occur in larger quantities in cow's milk than in human milk. In certain pathological conditions, however, the failure of the human infant to assimilate the salts of cow's milk produces serious disorders of nutrition and thereby becomes a factor in the production of marasmus and infantile atrophy. The common practice of adding lime water, sodium chlorid and other alkaline salts to cow's milk not only promotes the digestion and

absorption of casein, but also facilitates the absorption of its mineral salts, especially calcium.

The uses of mineral salts in the body are manifold. They are necessary for the growth and functional activity of all its cellular elements. They enter very largely into the construction of the bony framework which is growing so rapidly during the first year of life; for this purpose large quantities of calcium and phosphorus are especially necessary. They maintain the normal irritability of nerve and muscle elements; in this function calcium plays the most important physiological rôle. A partial calcium and phosphorus starvation produces not only abnormalities in the bony framework, but greatly exaggerated irritability of nerves and muscles. The causes that lead to calcium starvation, however, are not always to be found in the food, since MacCallum and others have shown that a diminished secretion of the parathyroid glands may be a factor in its production. The mineral salts also maintain the osmotic pressure which determines the flow of water to and from the fixed tissue cells producing a shrinking of the tissues on the one hand or a swelling (edema) on the other. They also regulate the acidity or alkalinity of the body fluids, a most important function, since upon the carefully adjusted reaction of the blood and other body media depends the normal functional activity of every cellular element in the body.

The mineral salts and their combinations are an essential part of the food of the infant. They must be presented not only in proper relative proportions, but in such a form that they can be readily assimilated. These objects cannot be accomplished during the early months of life in any other way except by the feeding of milk, preferably human milk whose saline constituents are held in such organic combinations that they are readily assimilated.

From the foregoing outline it is evident that all the food ingredients of milk are absolutely necessary to the health of the infant, and it is futile, therefore, to further discuss their relative importance. The normal infant has storehouses of protein, fat, carbohydrates, and salts always at hand, and has also a protective mechanism which enables it to substitute other materials for a time in case there be a partial starvation in any one of these food elements. But these storehouses may in time be exhausted and these protective mechanisms may fail, and then comes disaster to the infant in the form of some severe malnutrition, it matters not in which of the food elements there be a famine.

**Water.**—About 68 per cent. of the infant's body is composed of water and about 87 per cent. of its natural food (milk) is water. These facts indicate the important rôle that water plays in the physiological processes necessary to maintain the health and life of the infant. An infant requires four or five times as much water in proportion to body weight as an adult.

Water is the great solvent which brings into solution or suspension the food of the infant so as to present that food (the proteins, the carbohy-

drates, the fats, and the salts) in such a form that it can be readily cared for by the digestive organs. It carries the digested and assimilated food through the blood and lymph channels to every part of the body. This common carrier makes up about 78 per cent. of the blood and 96 per cent. of the lymph, and becomes the circulating media of the body, carrying the important elements of the blood and lymph to every cell in the body and carrying away from the cells to be excreted the waste materials of retrograde tissue metamorphosis. This excretion of body waste prevents autointoxication and is effected by the elimination of the water carrying this waste through the kidneys, the intestines, the skin, and the lungs. The discharge through all these avenues of excretion is much more active in infancy than later in life; this is especially true of the more active elimination through the skin of the infant, which is fourfold greater than in the adult. Martin H. Fischer has emphasized the fact that water is practically the only diuretic and diaphoretic we have; all other agents supposed to act in these ways do so indirectly by bringing to the kidneys or skin free water ready for excretion.

In the emergencies which disease produces nature takes advantage of these various channels for the rapid elimination of waste materials, and enormous quantities of poisonous fluids are carried off in a short time, especially through the skin and bowels. In these same emergencies the physician attempts to replenish the body fluids of the infant with pure water or physiological salt solution, so that vital processes may not suffer from the partial water famine which nature has created in her strenuous efforts to save the life of the child. Thus, in certain of these emergencies, water becomes a life-saver more important than food, stimulants, and all other agents. In the artificial feeding of infants an excess of water is not infrequently given. An infant should not be given more fluid than a breast-fed baby of the same age would obtain from its mother under normal conditions. Too much water in the food may cause dilatation of the stomach, indigestion, and consequent malnutrition.

**Digestive Ferments.**—Human milk contains a diastatic ferment which transforms starch into maltose and dextrose. Becamp was the first to find this ferment; it has since been demonstrated by a number of observers, and according to Spolverini may be developed in the milk of the cow and goat by feeding them on germinating barley. This would indicate that there is an important physiological purpose served by this ferment in the young human infant which is met in some other way in the young of the cow and the goat. The purpose served is perhaps to supplement the feeble digestive power for starch in the young human infant, and as the starch digestive capacity of the young of the cow and goat is greater it is not necessary to provide this ferment in their milk.

Lipase is a fat-splitting ferment which breaks down neutral fats into fatty acids and glycerin. It was isolated by Lussatti and Biolchini and is much more active in human than in cow's milk. This active fat-splitting ferment in human milk may account in part for the fact that young

infants are capable of digesting and assimilating much larger quantities of the fat of human milk than of cow's milk.

Arguing by analogy, one would think that nature, in developing a fat-splitting and a starch-digesting ferment in woman's milk, would also have developed a protein-digesting ferment, and this is very probably the case, although experimental evidence is not as yet sufficient to put this question beyond a doubt. Spolverini demonstrated the action of ferments which resembled the action of trypsin and pepsin, but Benoit could not agree with him.

**Alexins.**—Alexins are substances which have a bactericidal and globulicidal action. They are found in human milk in sufficient quantities to make it decidedly destructive to bacteria and other foreign cells. They are perhaps derived in part from both the serum of the blood and from the cells of the mammary gland. This property of human milk is perhaps one of the most important safeguards which the young breast-fed infant has against gastrointestinal diseases. The microorganisms, which are constantly being put into their mouths, on foreign objects, rarely produce any serious intestinal disturbance. It is also true that artificially fed infants when suffering from gastroenteritis will, as a rule, quickly recover when given breast milk, and it is probable that the bactericidal action of the milk is one of the factors that contributes to this result. The alexins are not absorbed in sufficient quantities from the milk to give to the blood and other body fluids of the infant any marked bactericidal qualities, so that the comparative immunity which breast-fed infants enjoy from certain acute infectious diseases is perhaps due to other substances, which pass from the mother through the breast milk to the infant, such as antitoxins, agglutinins, and other antibodies. The immunity, either natural or acquired, which the mother or wet nurse has for certain infectious diseases is, in part at least, transferred to the nursing infant. The substances which confer that immunity are transferred through the milk to the infant. It follows therefore that the nursing infants are partially immune from those infections only to which their wet nurse is immune.

## CHAPTER XI

### HUMAN BREAST MILK IN ITS RELATIONS TO INFANT FEEDING

### COMPOSITION OF COLOSTRUM AND HUMAN MILK

Colostrum is the secretion of the human breasts which immediately precedes the formation of breast milk. About the fourth month of pregnancy the breasts commence to secrete in scanty amounts a yellowish, sticky fluid called colostrum. This secretion, which can be squeezed out of the breasts in slightly increasing quantities from this time until the birth of the child, is present in sufficient quantities during the first few days after birth to act as a laxative and serve nutritional purposes.

Colostrum differs from normal milk in its physical and chemical properties. It is more alkaline in reaction, more yellow in color, is not so sweet, and has a higher specific gravity, 1.040. It is richer in fat and soluble proteins and poorer in casein. Under the microscope it shows peculiar large characteristic corpuscles which are filled with fat globules of varying sizes. On the removal of this fat by ether the corpuscle shows a large nucleus. These corpuscles are from five to ten times the size of the human blood corpuscle. Leukocytes and pavement epithelium are also present in addition to a large number of fat globules similar in size to those found in normal human milk. The composition of colostrum is shown in the following table from Camerer and Söldner:

| | |
|---|---|
| Water | 86.70 |
| Proteins | 3.07 |
| Fat | 3.34 |
| Milk sugar | 5.27 |
| Ash | 0.40 |

Under the stimulus of nursing the colostrum gradually gives way to the normal milk secretion, which may be established as early as the third day, but is sometimes more or less delayed until the fifth or sixth day.

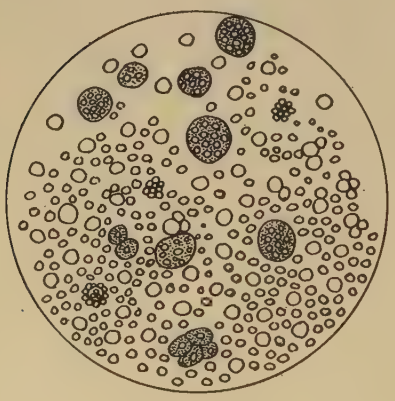


Fig. 17.—Colostrum.

Fig. 18.—Woman's Milk.

Human milk is a bluish white, sweet fluid which, as shown by Kerley and others, is faintly acid to phenolphthalein, but is amphoteric or neutral to litmus paper. Its specific gravity varies from 1.027 to 1.035. Microscopically the fat globules, which vary in size from that of a red to a white blood corpuscle, are held in more or less satisfactory emulsion. Epithelial cells, colostrum and pus corpuscles which are occasionally found are to be regarded as foreign elements and as indications pointing to the deterioration or contamination of the milk.

## HOW TO DETERMINE THE WHOLESOMENESS OF MILK

The fundamental characteristics which differentiate the milk of different species were dwelt upon in the previous chapter, and in these important facts we learn why it is so necessary to the nutritional demands of the infant that it should have all the breast milk it can possibly get under the existing circumstances. This principle in infant feeding is all-important since about two-thirds of the mothers of our land are unable to furnish their infants with a milk, which in quantity and quality is equal to the nutritional demands of the first nine months of life. Among the wealthy class it is rather unusual to find a mother who can supply breast milk to meet the demands of her infant. Among the poor the woman has to work the greater portion of the day in unhygienic surroundings, live on improper and perhaps insufficient food and be separated from her infant many hours at a time, which interferes with her furnishing normal milk at proper intervals throughout the full period of lactation. It is largely, therefore, among the middle class that we find women who are not only willing but who are physically able to fully nourish their infants for a proper length of time. In America, perhaps more than in other countries, the social conditions are unfavorable to the production of mothers capable of performing this most important function. At any rate, we are led to infer from published observations that supplemental and full artificial feeding are perhaps more commonly necessary in this country than in some European countries.

In approaching the subject of breast-feeding it is important that the physician should understand that there are ofttimes abnormal variations in the quantity and composition of an otherwise good milk, which may make it temporarily insufficient or unwholesome for the infant. It is plainly evident therefore that it would be worse than folly for the physician to infer that such a milk was not suited to the nutritional demands of the individual infant until a more or less prolonged trial of the milk, its chemical composition or the conditions under which it is produced have demonstrated that it is an unsuitable food. The physician should have in mind certain facts which will help him in determining the wholesomeness of an individual breast milk in its relation to the nutritional demands of an individual infant, and along these lines the following observations are important:

1. If a breast-fed baby is well nourished and gaining in weight, all indications pointing to the unwholesomeness of its food, such as colic and indigestion, are of minor importance and suggest *not* that the breast milk be discontinued, but that it be modified by regulating the diet and general hygiene of the mother.

2. If a breast-fed baby is poorly nourished and losing in weight, with no evidence on the part of the infant that the milk is producing gastro-intestinal disturbance, the indications are *not* that the breast milk should be discontinued, but that it be supplemented by artificial feeding at each

nursing and an effort made to increase the quantity and quality of the mother's milk by proper food and hygiene.

3. A poorly nourished breast-fed baby, losing in weight and suffering from chronic indigestion both on breast and mixed feedings, should be weaned or provided with another wet nurse. In these comparatively rare cases the individual breast milk is at fault even though chemistry may fail to find the defect.

If the question arises as to whether a mother's milk is a suitable food for the infant, the infant itself under the three fundamental rules above given is the best answer to this question. As supplemental evidence, however, an examination of the breast milk may be made to determine its quantity and also the relative and absolute quantity of its various ingredients. The quantity of the breast milk may be determined by weighing the baby before and after each nursing, and for practical purposes it may be assumed that an increase of an ounce in weight indicates that a fluid ounce of milk has been taken; that is to say, if the infant weighs five ounces more after a nursing it has taken five fluid ounces of milk; and it may also be assumed, for practical purposes, that infants between one and seven months of age should take in the average at each nursing one ounce more than they are months old. Holt gives the following table of the daily average quantity of milk taken by normal infants at different ages:

| | | |
|---|---|---|
| At the end of the 1st week | 10 to 16 oz. | (300 to 500 gm.) |
| During the 2d week | 13 to 18 oz. | (400 to 550 gm.) |
| During the 3d week | 14 to 24 oz. | (430 to 720 gm.) |
| During the 4th week | 16 to 26 oz. | (500 to 800 gm.) |
| From the 5th to the 13th week | 20 to 34 oz. | (600 to 1,030 gm.) |
| From the 4th to the 6th month | 24 to 38 oz. | (720 to 1,150 gm.) |
| From the 6th to the 9th month | 30 to 40 oz. | (900 to 1,200 gm.) |

The quality of the milk with reference to the amount of its various ingredients may be determined by a chemical analysis, but as the variations, especially in the fats and proteins, from day to day are very marked, it is generally necessary to make repeated examinations of the milk to arrive at its average composition. Milk analyses, therefore, because of their unreliability and the expense of making them, are not of sufficient practical value to warrant their use as a routine measure. In a breast-fed baby, suffering from well-marked scurvy, a professional chemist reported to me that the breast milk was absolutely normal. A change, however, to cow's milk in this case brought about a rapid cure. Simpler and less accurate methods for examining breast milk are used and the approximate results thus obtained may be of some value. For this purpose the physician may use Holt's apparatus, made by Eimer & Amend, of New York. Full directions for its use accompany each set of apparatus.

Holt gives the following table as the average composition of human breast milk:

| | Average per cent. | Common variations | Healthy per cent. |
|---|---|---|---|
| Fat | 4.00 | 3.00 to | 5.00 |
| Sugar | 7.00 | 6.00 to | 7.00 |
| Proteins | 1.50 | 1.00 to | 2.25 |
| Salts | 0.20 | 0.18 to | 0.25 |
| Water | 87.30 | 89.82 to | 85.50 |
| | 100.00 | 100.00 | 100.00 |

The sample of milk used for analysis should be a part of all the milk that can be taken from the breast. The physiological stimulus of sucking is not given by the breast pump, and the milk drawn in this way is therefore usually low in fat and protein. The usual examination of breast milk made from small samples drawn by the pump are misleading because they do not represent the same quality of milk which the infant receives from the breast.

## HOW TO MODIFY THE QUANTITY AND QUALITY OF MILK

In modifying human milk to suit the nutritional demands and digestive capacity of the infant it is most important that the general health of the mother should be carefully looked after. She should take a moderate amount of exercise in the open air, have sufficient sleep and rest and not be harassed by unnecessary petty household details, and above all the quantity and quality of her food should be carefully supervised to avoid gastrointestinal disturbance. She should eat a moderate quantity of fruits, vegetables, farinaceous and nitrogenous food, being always careful that the food selected is easily within her digestive capacity. Overeating, eating at irregular hours, drinking more than a very moderate amount of alcoholic stimulants and taking highly seasoned dishes, salads and foods difficult of digestion are to be carefully avoided. Constipation, when it exists, should be overcome by proper medicines and diet.

The quantity of the milk may be increased by improving the general health of the mother, by massage of the breasts and by certain foods, such as milk, gruels and the liquid malt extracts. The fat may be increased by meats, eggs, milk and the liquid malt extracts, and may be diminished by diminishing the quantity of these foods and substituting fruits, vegetables, bread and cereals. The proteins may be increased by rest and by giving an increased quantity of meat and eggs, and they may be lowered by exercise in the open air and by a diminished quantity of meat and eggs.

Menstruation, especially in neurotic mothers, may so change the character of the milk as to produce slight gastrointestinal indigestion, but these disturbances are rarely of enough importance to justify corrective measures of any kind.

Nervous impressions produced by great excitement, grief and fright may produce temporary changes in the breast milk sufficient to cause colic and indigestion in the infant, and prolonged nervous strain and worry

may be a very serious factor in diminishing the quantity and quality of the breast milk.

Arsenic, salicylic acid, alcohol, iodin, belladonna, opium, salts, iodids, bromids, cascara and senna when taken, may be eliminated through the milk in sufficient quantities to produce gastrointestinal indigestion or toxemia in the nursing infant.

Bacteria are occasionally eliminated in the milk, but the danger from this source is exceedingly slight. Tubercle bacilli, typhoid bacilli and streptococci are, however, sometimes found in the breast milk of mothers suffering from the constitutional diseases which these microorganisms produce. The acute infectious diseases when prolonged and severe, such as typhoid fever and scarlet fever, contraindicate nursing, not only because of the danger of infection to the child, but because of the depletion of the mother. Other less severe infections, such as measles, influenza and even diphtheria unless it be very severe, should not interrupt nursing. In these conditions if proper precautions are taken the infant, as a rule, escapes the disease, or, if not, contracts it in a very mild form from being partially protected by the antibodies which are eliminated through the milk, and the dangers thus incurred are much less than those incurred by weaning the young infant.

A syphilitic infant should not be permitted to nurse a nonsyphilitic wet nurse any more than a syphilitic wet nurse should be allowed to nurse a nonsyphilitic infant; in both instances the disease may be transferred. A congenitally syphilitic infant, however, may nurse its mother without danger of communicating the disease.

Active tuberculosis in the mother is an absolute contraindication to nursing. Mothers, however, who have recovered from active tuberculosis may under urgent conditions be allowed to nurse their offspring. The danger here, however, is rather to the mother than to the child, inasmuch as the drain upon her may so weaken her powers of resistance that a latent tuberculosis may develop into an active one.

General debility, malnutrition and pronounced anemia on the part of the mother, as a rule, contraindicate nursing unless the condition be a temporary one that can be readily removed by good hygiene and medication.

## CHAPTER XII

### BREAST FEEDING

### NORMAL BREAST FEEDING

It is a well-recognized fact that in healthy mothers the regular and vigorous nursing of the breasts by an infant strong enough to nurse is the most important single factor in stimulating the secreting glands of the breast to perform their normal physiological function; that is, to supply

an abundant quantity of normal milk. As Budin says, "the quantity of the milk varies with the demand." In the strong and vigorous under the stimulus of regular nursings the breast can in many instances be made to secrete sufficient milk to nourish two infants. The physician should therefore direct that the infant be put to the breast within four or five hours after birth, and during the first three days it is induced to nurse as vigorously as possible at intervals of four to six hours, dependent upon the condition of the mother. For three days even after a normal labor the mother is the prime consideration. It is most important that she should have proper rest and sleep undisturbed by regular nursings or by the care of her baby. The nursings during this time promote uterine contractions, clear the breasts of colostrum and hasten the establishment of the normal milk secretion on the third or fourth day of nursing. During these first days it is important that the infant should be given water to drink, either in the form of pure water, a milk-sugar solution, toast water, or a very weak solution of cow's milk, one of water to five of milk. A few teaspoonfuls of one of these mixtures given at intervals during the day will prevent loss of weight and furnish water for washing out the gastrointestinal canal, the kidneys, and other excretory organs. If the milk secretion is not established by the third day the milk mixture may be given in larger quantities until the delayed milk secretion is established. After the third or fourth day, with the mother thoroughly convalescent, regular nursings should be commenced. The baby should then be nursed every two hours from seven A. M. to nine P. M., and during the night should never be awakened for a feeding or put to the breast oftener than every four hours. In putting the infant to the breast the nipple should be worked out so that the child can get a firm grasp on the entire nipple. It should be allowed to nurse in the average fifteen minutes. Where the milk supply, however, is insufficient the time may be prolonged to twenty minutes, but during this time the child should not be allowed to lie with the nipple in its mouth, but should be kept nursing. When the baby is removed from the breast the nipple should be cleansed with water or boracic acid solution, and if there be any tenderness about the nipple it should be washed in a 50 per cent. alcohol solution and dusted with dermatol to harden and heal it. If there be erosions or fissures about the nipple these should be treated after each nursing with a 1 or 2 per cent. solution of nitrate of silver and the baby for a time be made to nurse through a nipple shield. Any disease of the nipple should receive prompt and careful treatment, as it may lead to caking of the breast, mastitis, or even abscess. The latter would be not only painful and interfere with the general health of the mother, but would cause the permanent loss of that breast to the infant. When, therefore, one of the breasts becomes swollen, hard and tender, accompanied possibly by fever, the baby should not be allowed to nurse from that breast until the inflammatory conditions have subsided, and the mother should be confined to bed for a few days with hot compresses of 20 per cent. alcohol, boracic acid, or some other antiseptic held over the breast in such a manner as to lift and support

it. During this time a breast pump may be used from time to time to relieve the engorgement, and under this treatment convalescence, as a rule, quickly results, the breast is restored to its normal condition, and the baby may again be allowed to nurse both breasts. In the event that such an emergency temporarily diminishes the milk supply of the infant so that it is insufficiently nourished it may be given, following every other nursing, a sufficient quantity of modified milk to make up the deficiency.

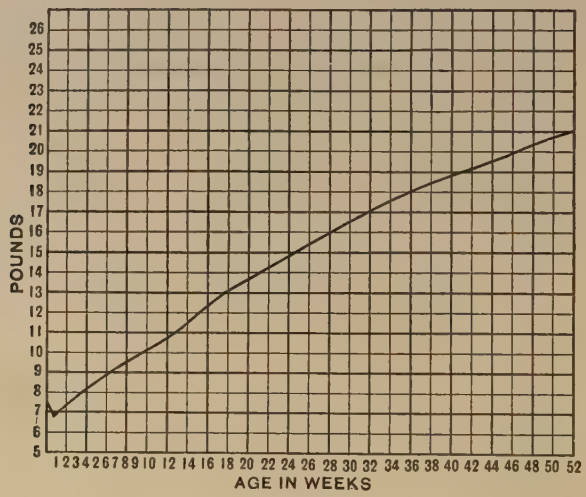



Fig. 19.—Weight Chart of Breast-fed Infant.

The intervals between the feedings are increased to two and a half hours when the infant is two months old, to three hours when it is four months old, and to four hours when it is nine months old, and during all of this time the baby must be fed at regular intervals, awakened on the stroke of the clock during the day and made to go as long as possible without nursing between ten in the evening and six in the morning. From the third to the fifth month one nursing at night should be sufficient, and as soon after the fifth month as possible the baby should be trained to go without nursing from ten at night until six in the morning. It is remarkable how a normal baby fed on normal milk will adapt itself to the regular hours of nursing, and in such infants the question of sleeping through the night is absolutely a matter of training. In a baby eight or nine months of age, trained to sleep the night through, the habit becomes so firmly fixed that it is rarely disturbed even by illness.

## MIXED FEEDING

When it has been determined that the breast milk is insufficient, mixed feedings should be resorted to; that is to say, the infant is to be given the breast at regular intervals, followed at every feeding by a sufficient quantity of modified milk to make up the deficiency. The success of this method of

feeding depends upon the following facts: First, frequent nursings will stimulate the secretion of milk and give the baby all the breast milk it can possibly obtain. Second, supplementing every nursing with the bottle, instead of nursing at one time and giving the bottle at another, insures sufficient nourishment at regular intervals, as the infant has the option of emptying the bottle or not after every nursing; while by the alternate nursing method, breast at one time and bottle at another, it may be a question with the infant of a feast and a famine. If the breast milk is insufficient it would be starved at alternate feedings. Third, and perhaps more important than all, is the fact that cow's milk is more easily digested when it is mixed in the stomach of the infant with breast milk. This is perhaps due to the more active ferments in human milk, which assist in the digestion of the starches, fat and proteins of cow's milk. At any rate, it is a fact which clinical experience has amply demonstrated that cow's milk is more easily cared for by the infantile digestive organs when it is mixed with human milk. The importance of mixed feeding has never been fully appreciated by the medical profession, and of all countries America is the one in which mixed feeding should be insisted upon, because here more than in European countries are we called upon to supplement mother's milk by artificial food. Mixed feeding, therefore, in the sense here outlined, is one of the most valuable expedients we have for getting good nutritional results in the feeding of infants.

Before dismissing the subject of mixed feeding I wish also to speak of the value of this method among the poor, where, because of their location and surroundings, they have to depend upon artificial infant foods to supplement nursing during the summer months. During these hot months these artificial foods, such as condensed milk, malted milk and Nestlé's food, are life-savers to this class of our population. It is absolutely impossible for city authorities to arrange that the poor of the city shall have the proper medical attention, and be furnished with a clean and properly modified milk, with the facilities for caring for the same. All of these things are absolutely necessary to insure the success of artificial feeding with modified milk. It becomes necessary therefore for the poor of our cities, in the vast majority of instances where the breast milk is insufficient, to resort to cheap, easily prepared and easily cared for artificial foods to supplement the breast feedings. In such instances and under such conditions it is to be strongly recommended that these artificial foods should follow the breast feedings. The breast milk in this form of artificial feeding will in large part prevent the scurvy, rickets, and other malnutritions that commonly follow the *long-continued* exclusive use of these proprietary foods. This method of mixed feeding with proprietary foods has been successful in my hands; I have utilized it in my dispensary work, and oft-times in my private practice, where the conditions were such that modified milk could not be safely used.

## WEANING

The question of weaning an infant is altogether an individual one, depending upon the individual conditions which one has to face in each instance. On general principles, however, it may be stated that the infant from birth should be accustomed to taking a little water in addition to breast milk, not that this water is necessary after the milk secretion is established, but that it is a good thing to accustom the baby to taking some other fluid than breast milk and to take it in a different way, either from the spoon or from a bottle. This, as a rule, obviates the difficulty which is occasionally encountered of starving the baby into taking artificial food. It is also a good practice even in perfectly nourished breast-fed babies, after the third or fourth month, to give them one bottle of modified milk as a substitute for one nursing in the twenty-four hours. This serves the double purpose of educating the infantile digestive organs to the digestion of cow's milk and of giving the nursing mother the opportunity of getting away from home and its duties for a brief period during the day. This relieves the mother of a certain amount of nervous strain and promotes her health and strength.

As time goes on in a perfectly normal breast-fed baby the number of modified milk feedings is to be increased, so that at eight or nine months the child is to have at least two feedings of modified milk in twenty-four hours. Within the next three months the number of feedings of modified milk is gradually increased until the infant at one year of age is weaned. The time of weaning, however, even in a normal child, may vary with the season of the year and with the health of the mother. If the birthday of the child comes during the hot months of summer it may perhaps be well to continue to give the child a few feedings of breast milk each day until it is thirteen or fourteen months of age, as this would enable the physician to utilize the breast milk in the emergency of any acute illness on the part of the gastrointestinal canal. In any event, whatever the conditions may be, it is much better, if possible, to wean the child gradually, letting it have the advantage of all the breast milk it can get for the first seven or eight months, and then during the next four months slowly educate its digestive organs to the digestion of cow's milk. Sudden weaning is justified only where acute conditions of ill health on the part of the mother make it absolutely necessary. Weaning during the hot months of summer is to be avoided if possible, and this is perhaps always possible under the method of mixed feeding outlined above. It is, of course, not always possible to solely consider the interests of the infant as to the time of weaning. The mother's health may demand that the child be weaned as early as the second, third or fourth months, but in such instances the method of mixed feeding given above should be used.

## THE WET NURSE

The conditions in America are such that the wet nurse is not as commonly used for breast-feeding the infant as in Europe. This is because the class of women who are willing to undertake this service are morally and physically of a much lower type, and yet it is possible to secure a more or less satisfactory wet nurse when the emergency arises which demands one. The wet nurse may be necessary to start the infant when the mother's milk cannot be used. This is especially true in premature infants or in malnourished infants born of tuberculous or otherwise diseased parents. It is also sometimes absolutely necessary following acute gastrointestinal diseases in the bottle-fed infant when other foods cannot be found upon which the infant can thrive. The physician must always bear in mind that a premature or malnourished infant that is doing badly under artificial feeding will probably continue to grow worse in spite of changes in artificial foods, and that such an infant may be expected to improve under good wet nursing. Wet nursing is to be preferred to artificial feeding in every such instance. Our knowledge of artificial feeding, however, has been so much improved of late that in the great majority of instances an infant can be successfully fed, supplying all its nutritional demands. This fact, together with the fact that satisfactory wet nurses are difficult to find and that they are undesirable members of a household, decides the mother in most instances to undertake the slight additional risk of artificially feeding her infant rather than undergo the expense and suffer the annoyance which the installation of a wet nurse would entail. It is therefore the important duty of the physician to determine in individual cases when a wet nurse is necessary.

**Selection of a Wet Nurse.**—A wet nurse must be free from all signs of tuberculosis, syphilis, and other chronic diseases. She should be in good physical condition, presenting the appearance of a woman capable of supplying sufficient milk to an infant without injury to her own health. She should also be comparatively young, preferably a primipara, and her baby should be over one month and less than seven months of age. Her own infant is the best indication of her fitness for wet nursing; it should show by its physical development that it has obtained a sufficient quantity of breast milk to supply its nutritional demands. There must also be a distinct understanding with the wet nurse before she is employed that she will conform to the rules of diet and hygiene necessary to produce the best results in furnishing a milk suitable to the nutritional demands of the infant she is to serve.

## CHAPTER XIII

### FOOD MATERIALS USED IN THE ARTIFICIAL FEEDING OF INFANTS

### COW'S MILK

**Fresh Cow's Milk.**—Cow's milk is the food almost universally recommended for the artificial feeding of infants.

The most important property of good cow's milk is cleanliness. There is little or no difficulty in obtaining clean milk in the country and in small towns, provided the ordinary rules of stable hygiene and personal cleanliness in the handling of the milk are observed. In large cities the problem is a very different one, since the milk has to be transported long distances, pass through many hands and the time between the milking and the consumption of the milk is greatly increased.

The great superiority of clean raw milk as an infant food over all other artificial foods has been so universally recognized in recent years that all of our large cities have made most strenuous efforts to obtain clean raw milk. The pioneer methods employed by the Walker-Gordon laboratories under the advice of Rotch, and the successful movement of Coit by which he was able to put upon the market a clean milk under the name of "certified milk," are largely responsible for the methods now employed in furnishing clean milk to our large cities. These movements have resulted in the establishment of model dairy farms so located that the rapid transportation of milk from the country to the city is possible. In the management of these farms the following conditions are necessary:—The cows must be healthy and free especially from tuberculosis; the stables must be clean and well ventilated and the barnyards free from manure and kept in as sanitary a condition as possible; the water supply used for dairy purposes should be pure; the food of the cows should be free from ensilage, strongly flavored weeds and distillery and brewery slops. The cows should be kept clean by daily grooming and the milking should be done with clean hands from clean udders into clean pails, using every precaution to prevent the early contamination of the milk. The first few strains of milk from each udder should be discarded. The milk should be immediately removed to a separate building, where it is rapidly cooled, bottled, and then placed in a refrigerator until it starts on its journey to the city. In the carrying of the milk to its destination it should be kept cool and should reach the consumer with a bacterial content not above 10,000 to the c. c. Milk furnished under these conditions may be fed in a raw state with safety to the infant.

The health boards and medical societies of our cities, following the initiative of Coit, have selected a board of chemists and bacteriologists whose duty it is to make frequent examinations of this milk as it is delivered to the consumer. These examinations determine the bacterial con-

tent of the milk, the amount of butter fat it contains, the relative percentage of its various ingredients, and the presence or absence of chemical preservatives and other foreign matter. When the milk of the individual dealer *always* conforms to the standards set by the medical boards he is furnished with labels containing the words "certified milk." These labels when placed upon the bottles are a guarantee to the consumer that the contained milk conforms in all respects to the standards laid down by the medical authorities.

The "certified milk" which is sold in nearly all of our large cities conforms to the following standards: First, freedom from pathogenic bacteria; second, a bacterial content not exceeding 10,000 to the c. c.; third, freedom from dirt and other foreign organic matter; fourth, freedom from chemical preservatives; fifth, a constant nutritive value with about 4 per cent. of fat and a proper percentage of proteins and carbohydrates.

It is plainly evident that milk of this character constantly supervised by competent chemists and bacteriologists is a great boon to every large city, but it is also evident that the production of this class of milk requires an unusual outlay of money on the part of the dairymen and must therefore be sold at a price that is absolutely prohibitive to the poor of our cities. For this reason in most of our large cities a second grade of milk is furnished by the same dairies and under the supervision of the same board of milk inspectors. The commercial name of this milk is "inspected milk" and it bears such a label testifying to its relative cleanliness. "Inspected milk" differs from "certified milk" chiefly in the standards required for its bacterial content. During the winter months it must not contain more than 60,000 bacteria to the c. c., and during the summer months not more than 100,000 to the c. c. In other bacteriological and chemical standards it is on the same plane as "certified milk," and sells a few cents cheaper by the quart.

**Sterilized Milk.**—But notwithstanding the efforts of health boards and medical societies it is impossible to put upon the market in large cities a clean milk at a price within the reach of the poor. The "certified and inspected milks" can be utilized only in the feeding of a comparatively small percentage of the infant population of our large cities. For this reason sterilization and pasteurization of milk still remain most important life-saving measures in the feeding of infants during the summer months, when, because of the heat, milk contamination increases rapidly. It must be remembered, however, that unclean milk—that is, milk that contains a large number of microorganisms—has undergone fermentative changes which injure its nutritive value and which may have produced poisonous, irritating bodies which make it a dangerous food for infants. Milk thus contaminated cannot be made wholesome by sterilization, so that when pasteurization or sterilization are resorted to as a means of preventing further bacterial contamination it is necessary to start with as clean a milk as possible.

Cow's milk may be sterilized by heating to 212° F., or 100° C., for twenty minutes. This produces what is ordinarily termed sterilized milk.

The heat destroys all the developed bacteria, but does not destroy the spores, and the milk is therefore not absolutely sterile, since these spores, after a time, may develop bacteria. The sporulated bacteria, however, are not of enough importance to justify the further application of heat. Sterilization of milk may be accomplished by means of the Arnold Steam Sterilizer, or by placing the milk bottles in boiling water for twenty minutes. The latter process is of great practical value among the poor of our large cities because of its simplicity and cheapness.

The advantages derived from sterilizing milk are as follows: It results in a loss of acidity on the part of the milk, which causes a retardation in rennin coagulation and thereby causes the casein to be precipitated in finer flakes so that it is more readily acted upon by digestive ferments; large casein curds never form in this milk. The fermentative processes are stopped and the milk is not further contaminated by bacteria. This is the prime object in the sterilization of milk, and greatly reduces the dangers of milk poisoning. Sterilization is the cheapest way of preserving milk. In fact, it is the only practical way by which it may be kept wholesome for use among the children of the poor in our large cities. These people cannot afford and cannot care for clean, raw milk. So with them the only available safe substitute in hot weather for condensed milk and the patent milk foods is sterilized milk. With infants capable of digesting sterilized milk it serves nutritional purposes much better than proprietary foods.

The disadvantages which are said to result from sterilizing milk are as follows: Decomposition of nuclein; separation of phosphorus from its organic union; partial coagulation of soluble proteins; partial destruction of the fat emulsion; increased difficulty in the digestion of casein; partial precipitation of citric acid as an insoluble calcium citrate; partial conversion of milk sugar into caramel; partial separation of the lime salts from their combination with calcium, thereby rendering them less easily absorbed; partial loss of carbonic acid, oxygen and nitrogen, which are expelled by heating; complete destruction of ferments, alexins, agglutinins and other live principles in the milk. These changes represent a distinct nutritional loss to the infant in that the important ingredients of the milk are rendered somewhat less digestible and assimilable. Sterilized milk also has a tendency to produce constipation with its resultant intestinal intoxications.

**Pasteurized Milk.**—When the medical profession awoke to the disadvantages of sterilized milk it began to experiment with lower temperatures, hoping in that way to destroy the developed bacteria without producing important chemical and biological changes in the milk. Koplik suggested the heating of milk for infant feeding at a lower temperature and Monti recommended a temperature of 180° F., and this process, in contradistinction to sterilization, was called pasteurization. Freeman has done very valuable work in popularizing a still lower temperature of pasteurizing which kills the greater portion of the developed bacteria and yet produces no serious chemical or biological changes in the milk. This process is very generally

used in this country, especially in our large cities during the summer months, and when properly used is a most important life-saving measure.

The Freeman Pasteurizer may be used for this purpose, and Freeman's conclusions are as follows: First, milk for infant feeding should be pasteurized so as not to interfere with its biological properties or chemical composition, but at a sufficient temperature to destroy the bulk of the bacteria present, including the tubercle bacilli. Second, a temperature of 140° F. (60° C.) continued for forty minutes would seem to fulfil these indications. Freeman also says the question "concerning the effect of heat on ferments has been carefully worked out by Hippius. The salol-splitting ferment found only in mother's milk was weakened by a temperature of 131° F. (55° C.) and destroyed by 149° F. (65° C.), while the amylolytic fer-

FIG. 20.—FREEMAN'S PASTEURIZER.
*A*, Bottles in position for heating; *B*, method of cooling.

ment found only in mother's milk was weakened by a temperature of 158° F. (70° C.) and destroyed by 167° F. (75° C.)."

It seems evident therefore that the pasteurization of milk when carefully done may serve the purpose of checking the fermentative processes without materially changing its chemical composition or biological properties, and clinical experience strengthens this opinion since milk pasteurized at low temperatures may be fed for a long time with practically the same results as are obtained from fresh, clean raw milk. Pasteurized milk, however, requires time, care and intelligence in its preparation, and also requires a subsequent refrigerator temperature to prevent bacterial contamination. It is therefore not commonly available for use among the poor of our large cities. It has, however, a large field of usefulness among those city dwellers who have the time and intelligence to prepare it and the facilities to care for it after its preparation, since in our large cities even the best available milk during the hot summer months is rendered safer by pasteurization.

**Peptonized Milk.**—Milk may be partially or wholly peptonized for the purpose of feeding premature infants, malnourished infants and those suffering from acute or chronic gastrointestinal disorders. In the handling of this class of infants there is unquestionably a field for the use of pep-

tonized milk. Many infants with feeble digestive capacity are capable of digesting and assimilating modified, peptonized milk in sufficient quantities to prevent rickets and other malnutritions which would follow if they were fed upon very weak milk mixtures. There are many children also who, after a long and severe gastrointestinal illness, do not recover for many months their capacity to digest unchanged milk. These children commonly thrive on peptonized milk, and it is sometimes even necessary to keep them on it until they reach the age when other foods may be added to their diet. In recommending peptonized milk it is also important to call attention to the fact that the child's digestive capacity may remain undeveloped if the peptonized milk be too long continued. As Chapin has forcibly noted the stomach of the infant must be gradually educated to digest milk, and this physiological process influences the anatomical and physiological development of the digestive organs. The physician must therefore not abuse the use of peptonized milk, nor on the other hand should he condemn the infant to rickets or other forms of malnutrition because of his fear that peptonized milk may weaken the infant's digestive capacity. Partially peptonized milk is prepared by using peptonizing tubes, one tube to a pint of milk, with a little bicarbonate of soda to prevent coagulation. The milk is peptonized at a temperature of 110° F. for ten or fifteen minutes and then immediately placed on ice to stop further peptonization. If after standing for several hours the peptonized milk is so bitter that the infant will not take it, sufficient cane sugar may be added for sweetening purposes. It is better not to boil the milk after peptonization, as this destroys the peptonizing ferments that have been added to the milk and produces other undesirable changes in the milk which have been previously referred to. It may be necessary in some instances to completely peptonize the milk. This is done in the same manner as above described except that the milk is kept warm and the peptonizing process continued for one and a half to two hours. This process makes the milk bitter and it is always necessary to overcome this bitter taste with cane sugar or saccharin.

**Buttermilk.**—Buttermilk as a food for infants deserves careful consideration. It has long been successfully used in Holland. In recent years the experience of physicians the world over has demonstrated that it may be a valuable substitute for cow's milk in infants suffering from various forms of gastrointestinal disturbance. Buttermilk used in infant feeding is commonly made from cream or milk that has soured naturally. The souring process in the milk, however, may be started or hastened by inoculation with sour milk or with lactic acid bacilli from a culture. The latter process, however, is not commonly practicable for the general practitioner. The composition of buttermilk varies. In the average, however, it contains about 1 per cent. of fat, 4 per cent. of sugar, and 3 per cent. of proteins. It has a food value of about 400 calories to the quart. It is commonly prepared for infant feeding as follows: To one quart of buttermilk are added two level tablespoonfuls of wheat flour and one level tablespoonful of cane

sugar. This mixture is, with constant vigorous stirring, slowly brought to the boiling point and kept there for twenty minutes, and then allowed to cool. The constant stirring prevents the coagulation of casein. Buttermilk prepared in this way has the same percentage of fat and protein as above given, but the carbohydrates have been increased to 10 per cent. and the food value of the mixture has been increased to 600 calories per quart. This buttermilk mixture, when considered from the standpoint of infant foods, contains a low percentage of fat and a comparatively high percentage of proteins and a very high percentage of carbohydrates. The casein is very finely divided, separated from its calcium base and appears in the form of the lactate of casein which cannot be acted upon by rennet, but which is readily digested by the intestinal ferments. The acidity varies in the neighborhood of 0.5 per cent. The chief value therefore which buttermilk has, when prepared as above described, lies in the comparatively large quantity of easily digested casein which it contains, the small amount of fat, and the large quantity of easily digested carbohydrate which substitutes for the fat in serving the nutritional demands of the body. Buttermilk, notwithstanding the fact that it may be used in the feeding of well infants for some months at a time without producing apparent nutritional disturbances, is an ill-balanced food mixture, not capable of satisfying the full nutritional demands of the rapidly growing infant. It is to be used therefore as a food for normal infants only when properly modified cow's milk cannot be obtained. Its real field of usefulness, however, is as a substitute for cow's milk in infants who are suffering from gastrointestinal disturbances and who are not capable of digesting cow's milk. It has been used with success in chronic and subacute gastroenteritis, infantile atrophy and acute gastrointestinal indigestion. This buttermilk mixture may be modified by the addition of boiled water to suit the age and digestive capacity of the infant.

**Finkelstein's Albumin Milk.**—Take one quart of boiled milk; after it is cool remove from the top the thick scum, then add to it a liquid rennet (1 ounce of commercial essence of pepsin) and allow it to coagulate for one hour at a temperature of 42° C. in a warm bath. Then thoroughly stir so as to break the coagulated casein into fine particles, and pour it into a bag of cheesecloth to drip for one hour. The casein is then removed from the bag and stirred into a pint of water and worked with a wooden spoon through a fine sieve. A pint of boiled buttermilk is then added and the whole mixture is again worked through a fine sieve until the casein is so finely broken up that it looks like ordinary milk. I have had a large and favorable experience during the last two years with Finkelstein's milk prepared as above directed. This mixture is said to contain 3 per cent. of protein, 2.5 per cent. of fat, 1.5 per cent. of sugar, and 0.5 per cent. of ash. It is therefore almost a sugar-free mixture very rich in casein and containing a fair percentage of fat. It is recommended in sugar intoxications. I have found it of special value in infants under two years of age suffering from chronic gastrointestinal indigestion. Under this food very commonly fever, diarrhea and toxic symptoms subside and the infant gains in strength,

stops losing weight and in some instances there is a slight gain. After this mixture has been used for two or three weeks I have found it advisable to begin the use of codliver oil; with this addition the infant commences to gain in weight, and thereafter thick cereal gruels in small quantities may be added. Later, as the infant becomes convalescent, ordinary modified milk formulas may be gradually substituted and the Finkelstein milk discontinued. During the use of this formula constipation must be combated by the use of milk of magnesia or some other laxative.

**Malt Soups.**—Malt soups, introduced by Keller (Breslau), will sometimes agree with infants that have failed to thrive on the ordinary milk formulas. These soups are rich in carbohydrates and weak in fat and protein. The excess of carbohydrate is, however, well borne, and Keller believes that in malt soups less protein is lost by intestinal fermentation and therefore more absorbed than in other milk foods. However this may be, the fact remains that the "malt soup" is of value in the feeding of some difficult cases. It is made as follows: One ounce (by weight) of wheat flour is rubbed up with enough cold milk to prevent lump forming and then mixed with 10 ounces of milk. This is heated slowly with constant stirring for twenty minutes and allowed to cool. In a separate vessel dissolve 3 ounces of one of the thick malt extracts (such as Maltine or Maltzyme) in 20 ounces of lukewarm water which contains 15 grains of potassium carbonate. This is then added to the milk and flour mixture and kept warm for two or three minutes and then rapidly heated with constant stirring for five minutes. Cool and the mixture is ready for use. It may be diluted, if necessary, for young infants.

**Skimmed Milk.**—Skimmed milk has only about 1 or 1½ per cent. of fat, but it has practically the same amount of sugar and protein as whole milk. It is of great value as a substitute for whole milk in infants suffering from gastrointestinal disturbances and in all other cases where there is an inability to digest the fat of cow's milk. Experience has demonstrated that skimmed milk has a wide field of usefulness as a temporary food in these cases. It should be modified by the addition of a carbohydrate mixture to suit the age of the infant, and if it is given for any length of time the carbohydrates should be added in excess to make up in caloric value for the loss in fat. As the infant regains its capacity for fat digestion the quantity of carbohydrates should be gradually diminished and the fat gradually increased so as to prevent nutritional disturbances which might result from the long-continued use of a food markedly deficient in fat.

## CARBOHYDRATES

Carbohydrates, including the sugars and starches, play a most important rôle in modified milk mixtures.

**Milk Sugar.**—Milk sugar has for many years been in high favor because it is a natural ingredient of milk and because it can, as a rule, be fed in sufficient quantities to produce good nutritional results without pro-

ducing gastrointestinal disturbances. Milk sugar, however, is more susceptible to fermentation and is not so readily assimilated as maltose and dextrin, and is therefore not an infrequent cause of intestinal fermentation and of the symptom group elsewhere spoken of under the term "sugar intoxication." On the whole, however, milk sugar is a safe and satisfactory form in which to administer carbohydrates. It is commonly used in 5 to 7 per cent. solutions dissolved in boiling water. But when fermentative changes occur in the intestinal canal the milk sugar should be suspected as the primary cause and the quantity of sugar diminished or a maltose-dextrin mixture substituted for the milk sugar.

**Cane Sugar.**—Cane sugar may also be used in the same manner as milk sugar, but because of its sweetness it cannot, as a rule, be used in large enough quantities to supply the carbohydrate demand of a modified milk mixture, and when given in excess commonly causes a fermentative diarrhea. Cane sugar, however, is very commonly used in small quantities to sweeten cereal decoctions. Cane sugar, like milk sugar, may also produce intestinal fermentation, and when this occurs it should be discontinued and a maltose-dextrin mixture substituted.

**Maltose.**—Maltose has for many years been considered one of the most valuable of infant foods in modifying milk formulas; but the German school in the last few years has called special attention to the value of this sugar as a substitute for milk and cane sugar in conditions of intestinal fermentation. It is more easily assimilated and more rapidly absorbed than lactose or saccharose and it may be taken therefore by the infant in larger quantities without producing sugar fermentation. The ferments which convert milk sugar and cane sugar occur exclusively in the intestinal canal, so that if this digestive process is not completed in the intestinal tract the partially converted sugars may be absorbed and produce a sugar intoxication. While, on the other hand, the ferment which converts maltose occurs not only in the intestinal canal, but in other parts of the body, so that, if partially converted maltose is absorbed, sugar intoxication is, as a rule, prevented by the further action of this ferment after this form of sugar has been absorbed, and this fact may partially explain the fact that the feeding of maltose rarely produces sugar in the urine.

Maltose is especially indicated in the feeding of very young and delicate infants, and in all cases where either milk or cane sugar has produced intestinal fermentation and sugar intoxication. In the feeding of maltose it has been found advisable to combine it with about equal parts of dextrin. In Germany, and later in this country, "Soxhlet's Nährzucker" (which contains maltose 52.44 per cent., dextrin 41.26 per cent., and sodium chlorid 2 per cent.) has been largely used. Mead's Dextri-Maltose (malt sugar), which contains about equal parts of dextrin and maltose, is a similar preparation which may be used instead of milk sugar or cane sugar for modifying milk mixtures. These dextri-maltose preparations have about the same caloric value as milk sugar, but, according to the experiments of Reuss, Grosz and others, their relative absorption per kilogram of body

weight as compared with milk sugar and cane sugar is as 7.7 to 3.1 grams. Mellin's food is a proprietary maltose dextrin mixture containing three or four times as much maltose as dextrin.

**Cereal Decoctions.**—Cereal decoctions such as barley, oatmeal and rice-water may be prepared by adding a slightly rounded tablespoonful (½ ounce) of barley flour, oatmeal or cracked rice to a pint of water and boiling for thirty or forty minutes, and then adding hot water to the mixtures to supply the loss from evaporation, so that the cereal decoctions will represent a pint of fluid for every tablespoonful of the cereal used. This makes approximately a 3 per cent. starch mixture. These cereal decoctions, especially barley water, have been strongly recommended by Jacobi for many decades and are now very generally used by the medical profession. They serve the double purpose of furnishing an easily digested carbohydrate food and of causing the casein to be precipitated in fine curds.

**Dextrinized Gruels.**—Dextrinized gruels are made by adding to one pint of a lukewarm cereal decoction, such as barley water, one tablespoonful of one of the thick malt extracts such as maltine or maltzyme, and, after five or ten minutes of stirring, the mixture is brought to a boil. Dextrinized gruels contain a variable amount of starch, dextrin and maltose. If the dextrinizing process lasts longer than thirty minutes all the starch is converted into dextrin and maltose. Experience has taught that a cereal decoction in which about one-half the starch is converted into dextrin and one-half into maltose, offers one of the best carbohydrate mixtures for infant feeding, and this result can be approximately obtained by dextrinizing the cereal decoction for ten minutes. Dextrinized gruels prepared in this way are especially adapted to furnish young and delicate infants with a carbohydrate food which is more easily digested and assimilated than the unmalted cereal decoctions. The old-fashioned "flour ball" is prepared by tying in a cloth a ball of wheat flour four or five inches in diameter, suspending it in boiling water five or six hours and then uncovering and drying the flour. The starch by this process is partly converted into dextrin. One tablespoonful of this partially dextrinized flour when mixed and boiled in one pint of water makes a very good carbohydrate mixture for modifying cow's milk.

**Condensed Milk.**—Condensed milk is prepared by evaporating cow's milk about one-fourth in volume. It is preserved by the addition of considerable quantities of cane sugar—five or six ounces to the pint.

The composition of condensed milk is shown in the following table from Holt:

| | Condensed Milk | With 6 Parts of Water Added | With 12 Parts of Water Added | With 18 Parts of Water Added |
|---|---|---|---|---|
| | Per Cent. | Per Cent. | Per Cent. | Per Cent. |
| Fat | 6.94 | 0.99 | 0.53 | 0.36 |
| Proteins | 8.43 | 1.20 | 0.65 | 0.44 |
| Sugar { Cane 40.44 / Milk 10.25 } | 50.69 | 7.23 | 3.90 | 2.67 |
| Salts | 1.39 | 0.17 | 0.10 | 0.07 |
| Water | 31.30 | 90.49 | 94.82 | 96.46 |

An examination of the percentages given in this table shows that condensed milk is not a properly balanced food for infant feeding. It is notably deficient in fats and proteins and contains too much sugar, so that infants fed exclusively upon this food for any length of time must suffer more or less seriously from nutritional disturbances. Condensed milk babies have feeble powers of resistance. Their teeth and bony skeleton are slowly and imperfectly developed and they are commonly fat, flabby, rachitic and anemic. But notwithstanding the fact that the long-continued use of condensed milk almost invariably produces more or less serious malnutrition it is a very valuable temporary food for infants under certain conditions. Its advantages are that it is easily digested, sterile, cheap, easily prepared, and easily cared for. It is therefore of great value among the poor of our large cities who cannot afford to buy clean cow's milk and who have not the facilities for keeping cow's milk clean and wholesome, even if it were furnished to them. Thousands of infants in our large cities are carried through the summer months on condensed milk who would have died from gastroenteric troubles if their mothers had been compelled to feed them upon such cow's milk as they could procure, and these rachitic, malnourished babies, who have passed through the crisis of their existence on condensed milk, as the cooler weather comes may gradually overcome these malnutritions by the addition to their diet of more wholesome food. In the care of condensed milk babies the physician, realizing the importance from a nutritional standpoint of substituting cow's milk for this food, too often makes the change more rapidly than the child's digestive capacity will permit and thereby adds a gastrointestinal disturbance to the malnutrition. When it is practicable these infants should have cow's milk added very gradually to the condensed milk mixture so that it may in time gradually displace the condensed milk mixture without disturbing the infant's digestive capacity.

## PROPRIETARY FOODS

**Nestlé's Food.**—Nestlé's food is one of the most easily digested of the proprietary milk foods. It is a valuable temporary substitute for cow's milk in infants suffering from acute gastrointestinal disturbances. Nestlé's food is also one of the worst of the proprietary foods for continuous administration. Its long-continued use as the sole article of diet very commonly produces severe forms of rickets and scurvy. Chittenden's analysis of Nestlé's food, prepared according to directions for infants of six months, shows that this mixture has only 0.81 per cent. of albuminoids and 0.36 per cent. of fat. This marked deficiency in fat and protein renders it quite unfit to serve the nutritional demands of the infant for any great length of time. When Nestlé's food is used as a substitute for milk mixtures in the gastrointestinal disturbances of infancy it should, as soon as the condition of the gastrointestinal canal will permit, have added to it small quantities of milk, and as the child convalesces the milk is slowly increased and the Nestlé's

food mixture diminished, until a modified milk formula replaces the Nestlé's food. When for any reason it is necessary to continue Nestlé's food for any length of time and cow's milk cannot be added to the mixture, the infant should be given as supplemental foods orange juice and codliver oil. By these additions it may be possible to prevent the scurvy and rickets which otherwise might follow.

**Malted Milk.**—Malted milk is a proprietary milk food which, like Nestlé's food and condensed milk, is very poor in fat and in the total quantity of solid matter it contains. It is therefore not to be recommended as an exclusive food for infants. It may, however, be used as a temporary substitute for milk as in traveling or where for any other reason cow's milk is not available. It is, like condensed milk, very extensively used among the poor of our large cities, because it is easily digested, easily prepared, and serves the purpose of tiding the infant over the hot summer months.

CHITTENDEN'S TABLE

*Composition of some infants foods as prepared for the nursing-bottle in comparison with mother's milk. Prepared according to directions for infants of six months.*

| | Mother's Milk | Malted Milk | Nestlé's Milk Food | Imperial Granum | Mellin's Food | Peptogenic Powder |
|---|---|---|---|---|---|---|
| Specific gravity | 1031 | 1025 | 1024 | 1025 | 1031 | 1032 |
| Water | 86.73 | 92.47 | 92.76 | 91.53 | 88.00 | 86.03 |
| Total solid matter | 13.26 | 7.43 | 7.24 | 8.47 | 12.00 | 13.97 |
| Inorganic salts | 0.20 | 0.29 | 0.13 | 0.34 | 0.47 | 0.26 |
| Total albuminoids | 2.00 | 1.15 | 0.81 | 2.15 | 2.62 | 2.09 |
| Soluble albuminoids | 2.00 | 1.15 | 0.36 | 1.67 | 2.62 | 2.09 |
| Insoluble albuminoids | 0 | trace | 0.45 | 0.48 | 0 | 0 |
| Fat | 4.13 | 0.68 | 0.36 | 1.54 | 2.89 | 4.38 |
| Milk sugar | 6.93 | 1.18 | 0.84 | 2.71 | 3.25 | 7.26 |
| Cane sugar | 0 | 0 | 2.57 | 0 | 0 | 0 |
| Maltose | 0 | 3.28 | trace | trace | 2.20 | 0 |
| Dextrin | 0 | 0.92 | 0.44 | 0.58 | 0.53 | 0 |
| Soluble starch | 0 | 0 | | | 0 | 0 |
| Starch | 0 | 0 | 1.99 | 1.22 | 0 | 0 |
| Reaction | alkaline | alkaline | alkaline | alkaline | alkaline | alkaline |

## ALBUMIN WATER

Albumin water is prepared by adding the white of an egg to 8 ounces of boiled water which may be slightly flavored with salt. This food is very largely used in acute gastrointestinal disturbances as a temporary substitute for cow's milk where the latter is contraindicated. The fact that it has been used in this way for many years and still remains a favorite with the medical profession is evidence that it is a valuable substitute food in the treatment of these conditions. In my experience, however, albumin water has not acted as kindly in these conditions as barley water and other substitutes that are used for the same purpose. It is valuable as a food in the regular diet of infants over eight months of age.

## MEAT PREPARATIONS

**Beef Juice.**—Fresh beef juice may be prepared by slightly singeing a beefsteak on both sides and then cutting it into small pieces about half an inch square and expressing the juice with a meat press. The singeing of the beefsteak answers the double purpose of partially sterilizing the steak and of giving to the expressed juice the flavor of cooked meat. When prepared in this way beef juice according to Holt has the following composition:

| | |
|---|---|
| Proteins | 2.90 per cent. |
| Fat | 0.60 per cent. |
| Extractives | 3.40 per cent. |
| Salts | 0.20 per cent. |
| Water | 92.90 per cent. |

It will thus be seen that beef juice contains approximately 3 per cent. of albumin. It is a valuable food for infants during the second year of life and may be given, if necessary, as a supplemental food during the last three months of the first year. It is also a valuable substitute food in all gastrointestinal disorders where milk is contraindicated. It is not only easily cared for by the digestive organs of the infant, but is a stimulant and food of great value in many cases where other foods are but poorly tolerated.

**Broths.**—Broths made from mutton, beef, and chicken to which a cereal has been added, in the proportion of one tablespoonful to the pint of broth, are valuable foods which enter into the dietary of the child during the second year of life. The plain animal broths made from beef and mutton, and from which the fat has been carefully skimmed, are useful substitute foods in the gastrointestinal disturbances of infancy where milk is contraindicated. These broths contain only about 1 per cent. of protein and therefore are of little value as foods, but they are stimulating and satisfying and may be used temporarily during the starving process in all conditions of infancy where it is necessary to temporarily withhold other foods.

# CHAPTER XIV

### ARTIFICIAL FEEDING

## VALUE OF PERCENTAGE FEEDING

The percentage composition of human milk is of great interest in that it gives the actual and relative amounts of fat, proteins, sugar and salts which the ideal infant food contains. There is no doubt but that the various ingredients of human milk are combined in actual and relative amounts to suit the digestive capacity and the nutritional demands of the human infant. Neither is there any doubt but that the formula of human milk would be our best guide in making an artificial food for infants if the

various ingredients of cow's milk resembled in every particular those of human milk. Both of these statements are self-evident facts, and upon them the American school of pediatricians has made percentage feeding the underlying principle in the artificial feeding of infants. By percentage feeding is meant that the percentages of the chief ingredients of an infant food shall be combined in proper proportions to meet the nutritional demands and suit the digestive capacity of the individual infant. In making an artificial food if one had only to consider the nutritional demands of an infant it would be an easy matter to write out a food formula conforming in the percentages of its principal ingredients to human milk, but as one has also to consider the food idiosyncrasies as well as the digestive and assimilative capacity of the individual infant for a food whose principal ingredients are derived from cow's milk and which are, as pointed out in another chapter, chemically, biologically, and physiologically different from the ingredients of human milk, it follows that the percentage formula of an infant food to meet these conditions must differ materially from woman's milk. An artificial food therefore, under the percentage or any other method of feeding, must be constructed primarily to conform to the digestive capacity of the infant, but in conforming to this standard the various ingredients of the food mixture should not differ in their relative proportions so widely from the composition of mother's milk as to produce a partial starvation in either protein, fat, carbohydrates or salts. If both the digestive capacity and the nutritional demands of the infant are considered in the making of an artificial food, more or less accurate relative percentage values must be maintained and experience has demonstrated that this is a practical and scientific method of feeding. The only danger in this method is that the physician may be so impressed with its accuracy that he may give more attention to his method than he does to the digestive capacity and nutritional demands of the individual infant.

## CALORIC STANDARD

The caloric value of woman's milk is of great interest in that it furnishes us with an accurate standard of the food value of the ideal infant food.

A calorie is the amount of heat required to raise the temperature of one kilogram of water 1° C.; this is the unit of value by which the heat- or energy-producing values of various foods are measured. The ingredients of milk have the following caloric values:

| | |
|---|---|
| 1 gram of protein will produce | 4.1 calories |
| 1 gram of fat will produce | 9.1 calories |
| 1 gram of sugar will produce | 4.1 calories |

Spiegelberg says: "From calculations made by different authors it can be said that a strong breast-fed baby in the first two months consumes daily about one-fifth of its body weight of milk. In the second quarter of the

year this diminishes to one-sixth or one-seventh. In the latter months of the year to one-eighth or one-ninth of the body weight. A two-months-old child therefore weighing 4,000 grams would take one-fifth of 4,000—that is to say, 800 grams each day. A seven-months-old child weighing 7,000 grams would take 1,000 grams."

These values accord with those given by Heubner, that infants consume daily during the first three months an amount of milk equal to 45 calories per pound of body weight; during the next quarter about 43 calories; during the third quarter about 38 calories, and during the last quarter of the first year of life about 34 calories. In general terms therefore one may say that an infant's food should have a caloric value of 40 for every pound of body weight. A twenty-pound infant would require 800 calories in twenty-four hours. In young infants one may have to slightly increase and in older infants slightly diminish these values.

To determine the number of calories in a food mixture multiply the number of ounces of each ingredient used in twenty-four hours by the fuel value of an ounce of this food, add the products and the sum will represent the caloric value of the food. The following table shows the energy quotients of different materials used in infant feeding:

| | |
|---|---|
| 1 qt. of whole milk contains | 690 calories |
| 1 qt. of ⅔ whole milk contains | 460 calories |
| 1 qt. of ½ whole milk contains | 350 calories |
| 1 qt. of ⅓ whole milk contains | 230 calories |
| 1 qt. of buttermilk contains | 410 calories |
| 1 qt. of ordinary skimmed milk contains | 410 calories |
| 1 qt. of fat-free skimmed milk contains | 320 calories |
| 1 oz. of fat-free skimmed milk contains | 10 calories |
| 1 oz. of ordinary skimmed milk contains | 13 calories |
| 1 oz. of whole milk contains | 21 calories |
| 1 oz. of buttermilk contains | 13 calories |
| 1 oz. of 7 per cent. top milk contains | 31 calories |
| 1 oz. of 10 per cent. top milk contains | 39 calories |
| 1 oz. of 16 per cent. cream contains | 54 calories |
| 1 oz. of carbohydrate contains | 120 calories |

From this table it will be easy to determine the caloric value of any food mixture. Take for example the following:

| | | |
|---|---|---|
| Top milk 7 per cent. fat | 16 oz. = | 31 × 16 = 496 calories |
| Sugar of milk | 2 oz. = | 120 × 2 = 240 calories |
| Water | 13 oz. | |
| | 31 oz. = | 736 calories |

The value of the above food formula is therefore 736 calories, and, since 40 calories are required for each pound of body weight, if we divide 736 by 40 we find that the above food mixture has the requisite number of calories to feed an 18-pound baby twenty-four hours.

The calorimetric standard of the German school is the only accurate method we possess for determining the amount of food an infant should take in twenty-four hours, and the importance of utilizing this standard

to prevent the over- or underfeeding of infants should be insisted upon, as by this standard alone can we be sure, in varying the different ingredients of an artificial food mixture to suit the digestive capacity of the infant, that we are giving it a food the energy equivalent of which will accurately satisfy all the demands of the body. The calorimetric standard, however, must not for a moment be considered as a method of feeding or as the sole or all-important principle upon which a baby food is to be constructed. It is simply a standard which prevents the over- or underfeeding of infants by whatever method we adopt. An infant may be fed the proper number of calories of an ill-balanced food, such as condensed milk and the various patent milk foods, and yet suffer serious nutritional disturbances because of the relatively low percentages of fats and proteins in these foods. This illustration emphasizes the fact that the proteins, fat and carbohydrates, apart from their caloric values, have definite and distinct purposes to serve in supplying the nutritional demands of the infant. It follows therefore that the percentage method of feeding, which gives to the infant the relative quantities of fat, carbohydrates, proteins and salts it needs, must ever remain the basis of scientific infant feeding, and that in using this method one should, to secure fairly accurate quantities, conform, within certain limits, to the calorimetric standard, and should, to prevent or correct gastrointestinal disturbances, change the percentages of the various food ingredients to conform to the digestive capacity and food idiosyncrasies of the individual infant.

## PRINCIPLES OF ARTIFICIAL FEEDING

Success in infant feeding depends not upon the particular method used, but upon the intelligence, the experience and the knowledge of the underlying principles of infant feeding which the physician possesses. Methods of infant feeding are to experienced physicians what tools are to artisans. As one artisan may be more skilled in the use of a certain tool than another, so one physician may be more skilled in the use of a certain method of feeding than another. The principles which underlie the art of infant feeding are, therefore, so much more important to success than are individual methods that one is justified in trying to so formulate these principles that they may form the nucleus around which the young physician may build the clinical experience which will enable him more intelligently to use whatever method of infant feeding he may choose.

The principles underlying the artificial feeding of infants do not and possibly never will constitute an exact science, since the capacities and idiosyncrasies of infants in the digestion and assimilation of protein, fat, carbohydrates and salts cannot be foretold. There must therefore always remain certain difficulties to be overcome by experimentation. These experiments, however, should be carried along certain lines derived from clinical experience. The following principles derived from this source will be of value in the artificial feeding of infants.

I. Clean milk is absolutely necessary to success in infant feeding. Unclean milk cannot by any process be made into a safe and wholesome infant food; if pasteurization is necessary clean milk must be used to start with. This principle of infant feeding is absolutely essential and is much more important to success than methods of modification. Cream is always much more contaminated with bacteria than milk of the same age, and commercial cream, which is from twelve to twenty-four hours older than milk, contains a bacterial content so large that it is unsafe for infant feeding. If it be necessary to increase the amount of fat in an infant's food this may be done by taking a certain percentage from the top of clean milk. In this way one obtains a clean, wholesome cream. The formulas for the making of cream mixtures which are so widely spread among the laity are the cause of no small part of the digestive disturbances which occur in infants. The milk used in infant feeding should come preferably from common cows (Holstein and Ayrshire) rather than from highly bred ones of the Alderney or Jersey variety. The milk from highly bred cows contains too high a percentage of fat and this fat is not as digestible as that found in the milk of the common cow.

II. The infant should be placed upon a food formula which contains about the requisite number of calories. If, however, it fails to thrive and the gastrointestinal conditions remain normal the number of calories (the strength or quantity of the food) should be increased. *An infant should never be allowed to starve because it fails to thrive on the number of calories which a mathematical calculation awards it.* On the other hand, if an infant has become ill on a food formula one of the first inquiries to make is as to whether it is being overfed in calories.

III. Overfeeding, not only in the number of calories but also in the *number of ounces* given, is the cause of many failures in infant feeding. It is most important therefore that the number of ounces taken by the individual infant should be adjusted to suit the capacity of its stomach. To overfeed in ounces given will cause gastric and intestinal disturbance or will result in dilatation of the stomach with chronic gastric indigestion which months of careful feeding are required to overcome. To give an infant all it will take is a very common practice and one fraught with the gravest dangers. The capacity of the stomach of the average newly born infant is about one ounce and in premature infants it is less. The stomach grows in size rapidly for the first four months, at which time its capacity is about 5 or 6 ounces. After the fifth month the stomach develops less rapidly, so that at ten months it should hold about 9 or 10 ounces. At one year of age the stomach should hold from 10 to 12 ounces. Under one year of age the total quantity of liquid given in twenty-four hours should not exceed 40 or 45 ounces. In under- or oversized infants the number of ounces given may be slightly increased or diminished to suit the weight of the child. Overfeeding, in the number of calories given, is also one of the most common causes of digestive disturbance in infancy. But under these conditions the overfeeding does not always refer to an excessive fuel value

of all of the ingredients of the food, but may be due to a great preponderance of the carbohydrates, fats or whey salts. The important fact to bear in mind is that infants may be made ill with a perfectly wholesome food if given in excessive quantities and the overfeeding may comprehend an excessive quantity of all or of any one of the ingredients of its food.

IV. Artificially fed infants should be given their food at regular intervals. This is absolutely necessary to obtain the best results. The infant must be fed not only regularly but the interval between the feedings must be carefully adjusted to suit its age, weight and digestive capacity. From the end of the first week to the end of the fourth week a two-hour interval should be maintained; during the second and third months a two-and-a-half-hour interval; from the third to the sixth month a three-hour interval, and thereafter a four-hour interval. These periods should be observed with the strictest regularity, between the hours of 6 A. M. and 10 P. M., until the child is five months of age, and between the hours of 10 P. M. and 6 A. M. a much longer interval should be observed. After the fifth month the night feeding should be discontinued. When eight months of age the infant should have but four feedings in twenty-four hours, beginning at 6 or 7 A. M. and finishing at 6 or 7 P. M.

V. Rest for the nervous system is a most important aid to infantile digestion. Infants should be taught to lie quietly without coddling and should live in quiet surroundings, so that they may get the full amount of sleep due them. The immature nervous system of the infant, when excited by outside causes, exerts a most profound influence over the digestive organs.

VI. Fresh air is perhaps the most important outside aid in improving the digestive capacity of the infant. As soon as possible, after the first month in winter and almost from birth in summer, it should be taught to live and sleep during the greater portion of the day in the open air. Northrup has laid great stress upon the fact that it is quite as important for success in infant feeding "to modify the baby" by rest and fresh air as it is to modify the food.

VII. The infant's food should contain fat, protein and carbohydrates in fairly accurate percentages; that is to say, these ingredients should be combined in such relative quantities as will best meet the nutritional demands of the infant. Should it be necessary in the treatment of digestive disturbances to change the food formula by markedly cutting down either the fat or the protein, the physician should be most careful to see that the permanent formula adopted during convalescence should return in fat and protein as nearly as possible to the original formula. The necessity for this lies in the fact that no other food ingredient can take the place of protein, and that while the carbohydrates may for a time partially substitute for fat, the continued use of very low fat percentages will in time produce serious nutritional disturbances. The partial starvation of infants in protein, fat, and salts results in rickets, scurvy, anemia and other malnutritions. These ingredients should therefore, within the limitations of the

child's digestive capacity, be gradually restored to the quantities which the age and weight of the child demand.

VIII. The fat of cow's milk is not as readily digested as that of human milk. It is therefore wise to place the percentages of fat in modified milk mixtures for young infants much lower than it is in human milk. Excess of fat is one of the most common early causes of indigestion. To correct this condition the first trial change to be made in the food formula is a reduction of fat (cream) and a corresponding increase, in calories, of the carbohydrates. The indications for this change are strengthened if the infant has the following symptoms: Habitual regurgitation, constipation with gray, dry stools, or loose movements with small, soft curds and an irritating urine with an ammoniacal odor.

IX. The casein of cow's milk may be a cause of indigestion. If, therefore, the reduction of fat fails to correct the trouble an effort should be made to prevent the coagulation of casein in the stomach, by adding to the food sodium citrate, alkalies or cereal decoctions, or perhaps by boiling the milk. If these measures fail the casein may be diminished and the whey proteins added to make up the deficiency. When the cause of the trouble is thus righted the fat and the protein may be slowly increased to the original formula. Casein indigestion is indicated by large, tough curds, putrid, loose, brownish, alkaline stools, fever and other constitutional symptoms.

X. The carbohydrates, including the sugars, are the most easily digested of the food ingredients of a modified milk mixture, and for this reason they are not uncommonly increased at the expense of fat and protein. Under such conditions a carbohydrate indigestion and sugar intoxication may result. This may be indicated by a watery, acid, nonputrid diarrhea which produces irritation of the buttocks and which is frequently associated with fever, severe constitutional symptoms, much gas formation and intestinal catarrh. When this occurs the sugars must be temporarily eliminated from the diet until these symptoms subside and then a different carbohydrate (sugar) should be added, preferably one that contains a large percentage of maltose and dextrin, since they are less liable to produce fermentation than any of the other sugars or starches.

## HOME MODIFICATION OF MILK

The home modification of milk is almost universally used for infant feeding. The term "home modification" carries with it no definite conditions for the modification of milk. It is simply a general term used to cover all methods by which milk is modified at home with the idea of making it more suitable to the digestive capacity and nutritional demands of the infant. Nearly every physician of experience has worked out for himself a plan for modifying milk which his clinical experience has taught him will serve his purposes better than any other that he has been able to find, and nearly every author offers his own plan for the home modification of milk by which a certain degree of accuracy in percentage feeding

may be obtained. This state of affairs proves that there is no single method which outranks all others. The object of all these methods is to give the physician certain rules of thumb by which he may make milk formulas containing definite percentages of protein, fat, carbohydrates, and salts. Nearly all of these methods of feeding are more or less complicated in that they are formulated upon the idea that very exact percentages of protein, fat, carbohydrates, and salts are necessary to obtain good results in infant feeding, and the complications of the various methods commonly defeat their very objects, in that the average physician will not take the time or trouble to work out by them the exact percentages of the ingredients of a modified milk mixture. While there can be no objection to accurate percentages in infant feeding, yet the experience of the world has demonstrated that these accurate percentages are not absolutely necessary to success, and that on the whole infants thrive just as well upon a milk mixture which is intelligently modified so that protein, fat, carbohydrates, and salts are found in fairly definite percentages and in such quantities that the infant will not suffer from a starvation of any one of these important ingredients. Largely as a result of the complicated methods now in vogue, most physicians commonly use whole milk and some diluent in the form of a carbohydrate food, and upon these simple mixtures the large percentage of our infantile population is now thriving. While the simplicity of the "whole milk" method of modifying food has made it popular, there is no question but that much better results can be obtained by a method which utilizes top milk as well as whole milk in the preparation of infant foods, in that in this way the

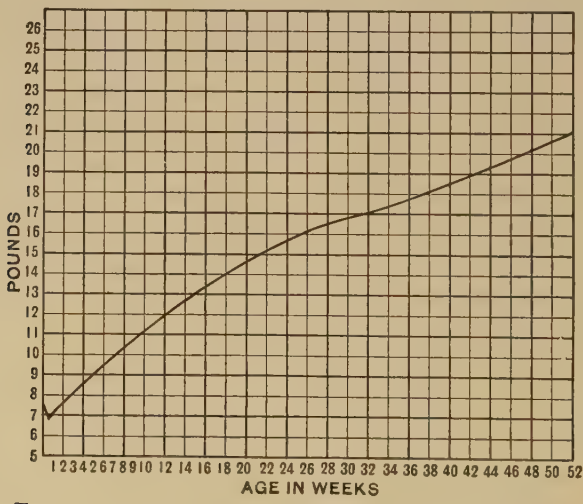



FIG. 21.—WEIGHT CHART OF ARTIFICIALLY FED INFANT.

fat percentages may be better adapted to the nutritional demands of the infant.

Percentage feeding may be greatly simplified and its efficacy, as I be-

lieve, not materially diminished by making all food formulas from three ingredients, viz.:

I. Whole milk, which to simplify computations one may assume contains 4 per cent. of fat, 4 per cent. of protein, and 4 per cent. of carbohydrate.

II. Top milk containing 7 per cent. cream, obtained by taking the top half of the milk after it has stood for two hours. This contains approximately 7 per cent. fat, 4 per cent. protein, and 4 per cent carbohydrate.

III. A carbohydrate solution made of sugars or starches containing one-half ounce of carbohydrate to the pint.

In infants under six months of age, because of the greater dilution of the milk, the 7 per cent. top milk should be used, so as not to get too low percentages of fat. In infants over six months of age whole milk may be used.

In making modified milk mixtures from these ingredients the physician should be guided by "the principles underlying the artificial feeding of infants," previously outlined. The following tables show how these ingredients may be combined in the production of an infant food which will answer all practical purposes, that is to say, will possess the proper number of calories, will contain the important ingredients, proteins, fat, and carbohydrates in such relative and absolute quantities that the nutritional and digestive demands of the infant will be satisfied quite as well as by more complicated mixtures in which the percentages of protein, fat, and carbohydrates are worked out to the fraction of a per cent.

Between the eighth and the twelfth month the food of the average well infant outlined in this table is to be supplemented by the addition of orange juice, raw egg albumin, thick cereal gruels, meat juice, and meat broths with a cereal admixture, but these foods, with the exception of orange

7 Per Cent. Top Milk Table

| Age | Estimated Weight | Ounces at Feeding | Interval of Feeding | Number of Feedings in 24 Hours | Total Quantity in 24 Hours | Whole Milk | Top Milk (7 per Cent.), Ozs. of | Milk Sugar, Ozs. of | Boiled Water, Ozs. of | Lime Water, Ozs. of | Percentage of Fat | Percentage of Protein | Percentage of Carbohydrate | Calories in Mixture | Calories Required |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 Wk.. | 7.5 | 2 | 2 | 10 | 20 | | 6 | 1 | 13 | 1 | 2 | 1 | 6 | 306 | 300 |
| 2 Wks.. | 7.5 | 2 | 2 | 10 | 20 | | 6 | 1 | 13 | 1 | 2 | 1 | 6 | 306 | 300 |
| 3 Wks.. | 8.0 | 2½ | 2 | 9 | 22½ | | 6½ | 1 | 15 | 1 | 2 | 1.1 | 5.6 | 321 | 320 |
| 4 Wks.. | 8.5 | 3 | 2½ | 8 | 24 | | 7 | 1⅛ | 16 | 1 | 2 | 1.1 | 5.7 | 352 | 340 |
| 2 Mo.. | 10.75 | 4 | 3 | 7 | 28 | | 9 | 1¼ | 18 | 1 | 2.2 | 1.3 | 5.5 | 429 | 430 |
| 3 Mo.. | 12.50 | 5 | 3 | 7 | 35 | | 10½ | 1½ | 23½ | 1 | 2.1 | 1.2 | 5.5 | 505 | 500 |
| 4 Mo.. | 14 | 5 | 3 | 7 | 35 | | 11½ | 1¾ | 22½ | 1 | 2.3 | 1.3 | 6.4 | 565 | 560 |
| 5 Mo.. | 15 | 6 | 3½ | 6 | 36 | | 13 | 1¾ | 22 | 1 | 2.5 | 1.5 | 6.5 | 603 | 600 |
| 6 Mo.. | 16 | 6½ | 3½ | 6 | 39 | | 15 | 1½ | 23 | 1 | 2.7 | 1.5 | 5.5 | 645 | 640 |

WHOLE MILK TABLE

| Age | Estimated Weight | Ounces at Feeding | Interval of Feeding. | Number of Feedings in 24 Hours | Total Quantity in 24 Hours | Whole Milk (4 per cent. Fat) | Top Milk (7 per Cent.), Ozs. of | Milk Sugar, Ozs. of | Boiled Water, Ozs. of | Lime Water, Ozs. of | Percentage of Fat | Percentage of Protein | Percentage of Carbohydrate | Calories in Mixture | Calories Required |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 Wk.... | 7.5 | 2 | 2 | 10 | 20 | 7 | | 1¼ | 12 | 1 | 1.4 | 1.4 | 7.5 | 307 | 300 |
| 2 Wks... | 7.5 | 2 | 2 | 10 | 20 | 7 | | 1¼ | 12 | 1 | 1.4 | 1.4 | 7.5 | 307 | 300 |
| 3 Wks... | 8 | 2½ | 2 | 9 | 22½ | 8 | | 1¼ | 13½ | 1 | 1.4 | 1.4 | 7 | 318 | 320 |
| 4 Wks... | 8.5 | 3 | 2½ | 8 | 24 | 9 | | 1¼ | 14 | 1 | 1.5 | 1.5 | 6.5 | 339 | 340 |
| 2 Mo.... | 10.75 | 4 | 3 | 7 | 28 | 14 | | 1¼ | 13 | 1 | 2 | 2 | 6 | 444 | 430 |
| 3 Mo.... | 12.50 | 5 | 3 | 7 | 35 | 19 | | 1 | 16 | 1 | 2.2 | 2.2 | 5.2 | 519 | 500 |
| 4 Mo.... | 14 | 5 | 3 | 7 | 35 | 21 | | 1 | 13 | 1 | 2.5 | 2.5 | 5.5 | 560 | 560 |
| 5 Mo.... | 15 | 6 | 3½ | 6 | 36 | 23 | | 1 | 11 | 1 | 2.6 | 2.6 | 5.5 | 603 | 600 |
| 6 Mo.... | 16 | 6½ | 3½ | 6 | 39 | 25 | | 1 | 13 | 1 | 2.6 | 2.6 | 5.3 | 645 | 640 |
| 7 Mo.... | 16.75 | 8 | 4 | 5 | 40 | 27 | | 1 | 13 | 1 | 2.6 | 2.6 | 5.1 | 687 | 670 |
| 8 Mo.... | 17.50 | 8 | 4 | 5 | 40 | 28 | | 1 | 12 | 1 | 2.8 | 2.8 | 5.3 | 708 | 700 |
| 9 Mo.... | 18.25 | 8 | 4 | 5 | 40 | 29 | | 1 | 10 | 1 | 2.9 | 2.9 | 5.4 | 729 | 730 |
| 10 Mo.... | 19 | 8 | 4 | 5 | 40 | 32 | | ¾ | 7 | 1 | 3.2 | 3.2 | 5.1 | 760 | 760 |
| 11 Mo.... | 20 | 10 | 4 | 4 | 40 | 35 | | ½ | 4 | 1 | 3.5 | 3.5 | 4.75 | 800 | 800 |
| 12 Mo.... | 21 | 10 | 4 | 4 | 40 | 40 | | 0 | 0 | 0 | 4 | 4 | 4 | 840 | 840 |

juice, should always precede or follow the bottle, which remains the chief part of the meal.

It may be noted by a glance at the whole-milk table that the fat percentages are somewhat lower and the protein and carbohydrate percentages somewhat higher than is commonly recommended by American authors. These changes from the routine formulas, however, are not objectionable, since they make the food more easily digested. The diminished amount of fat, which is rather difficult of digestion for the average infant, is made up by the increase in carbohydrates, which are very easily digested, and both of these changes facilitate protein digestion. The object of the table is to offer a method so simple that the busy physician can hold it in his mind and by it make modified milk mixtures which conform both to the percentage method and caloric standard.

**Carbohydrate Diluents.**—The carbohydrates in these milk formulas may be changed at the will of the physician, and in doing so it should be remembered that all carbohydrates used in infant feeding have practically the same food value. One ounce of milk sugar has the same caloric value as 1 ounce of cane sugar, wheat flour, barley flour, or oatmeal, and in making the carbohydrate diluent of an infant food they should all be used in the proportion of one-half ounce to the pint of water. In very young infants it is perhaps better to use dextrinized gruels made from barley or wheat. In older infants (after the third or fourth month) the unchanged cereal gruels made from barley, wheat, and oats answer quite as well as those which have been dextrinized. The carbohydrate diluents facilitate the digestion of protein. This action is perhaps purely mechanical in that the casein of

milk, when surrounded by a carbohydrate mixture, is precipitated in finer flocculæ and large curd formation is prevented.

**Alkaline Diluents.**—Experience has taught that the addition of alkalies to modified milk mixtures facilitates their digestion. The alkalies in most common use are lime water and bicarbonate of soda. The alkalies facilitate the digestion of casein by inhibiting the action of the rennet ferment, delaying the coagulation of casein and neutralizing the fermentation acids. Of the two alkaline diluents, lime water and bicarbonate of soda, lime water is more commonly used. It has the same action as the soda in preventing tough curd formation and acts more powerfully in stimulating the secretion of hydrochloric acid. Lime water should be used in the strength of 1 or 2 ounces to every 20 ounces of food, and bicarbonate of soda, 1 grain for every ounce of food. Chlorid of soda may be used instead of the bicarbonate in the same strength. If alkalies are used in too large quantities all action in the stomach on casein is suspended, and it passes uncoagulated into the small intestine. If this action of the alkalies is desired to prevent the coagulation of the milk in the stomach, 2 to 2½ grains of bicarbonate of soda should be used for each ounce of food. Citrate of soda is not an alkali, but when added to milk it decalcifies the casein, and prevents the action of the rennet. This decalcified casein forms with hydrochloric acid soft, friable flakes, in this way preventing the formation of tough curds. It may therefore be of considerable value in promoting the digestion of milk when there is a tendency to tough curd formation. One grain of sodium citrate for each ounce of milk in the food will insure the formation of soft curds instead of tough ones.

**Determination of Accurate Percentages.**—For those who desire to use food formulas in which the percentages of the important ingredients are more accurately determined, the following table, adapted by Southworth from that of J. F. Connors is given on page 140.

Southworth says: "The proteids have been calculated upon the basis of both 4 per cent. and 3.50 per cent. The former, 4 per cent., is for those who use round numbers to facilitate mental calculation of percentages. The latter, 3.50 per cent., which is the actual percentage of proteids in good average milk having 4 per cent. fat, is to enable the practitioner to determine readily the more exact amount of proteids in any given mixture. Either column may be used for the purpose of making a mixture of any desired percentages or in determining the percentages contained in any mixture of known proportions. To make up any desired percentage mixture (1) find in the one of the proteid columns determined upon the desired percentage, or that which is nearest it; (2) move in a horizontal line to the right until the desired percentage of fat is reached, or one which is nearest to it; (3) the heading of this fat column tells what kind of milk is to be used; (4) on the same line with the fat percentage at the right will be found the fraction showing the necessary proportions of this milk or top milk in the food mixtures to give the percentages selected, and beyond this will be found the number of parts of such milk or top milk and

## Key to Home Modification of Bottled Milk

| Per Cent. Proteids | | Per Cent. Fat | | | | | | | Per Ct. Sugar | Proportions of Milk and Diluent in Feeding Mixtures | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Figures Used in Mental Calculations. In Skimmed, Whole, or Top Milk, 4% Proteid | Figures Showing Actual Per centage. In Skimmed, Whole, or Top Milk, 3.50% Proteid | Skimmed Milk, Fat About 1% | Good Whole Milk, Fat About 4% | Top, 20 Ozs., Fat 6%, 1½ Times Whole Milk | Top 15 Ozs., Fat 8%, 2 Times Whole Milk | Top 11 Ozs., Fat 10%, 2½ Times Whole Milk | Top 9 Ozs., Fat 12%, 3 Times Whole Milk | Top 8 Ozs., Fat 14%, 3½ Times Whole Milk | Top 7 Ozs., Fat 16%, 4 Times Whole Milk | For Skimmed, Whole or Top Milk, 5% | Proportions of Milk | Parts Milk or Top Milk | Parts Diluent | Total Parts. |
| 0.25 | 0.22 | 0.06 | 0.25 | 0.37 | 0.50 | 0.62 | 0.75 | 0.87 | 1.00 | 0.31 | 1/16 | 1 | 15 | 16 |
| 0.33 | 0.29 | 0.08 | 0.33 | 0.50 | 0.67 | 0.83 | 1.00 | 1.17 | 1.33 | 0.42 | 1/12 | 1 | 11 | 12 |
| 0.50 | 0.44 | 0.13 | 0.50 | 0.75 | 1.00 | 1.25 | 1.50 | 1.75 | 2.00 | 0.62 | 1/8 | 1 | 7 | 8 |
| 0.57 | 0.50 | 0.14 | 0.57 | 0.86 | 1.14 | 1.43 | 1.71 | 2.00 | 2.30 | 0.70 | 1/7 | 1 | 6 | 7 |
| 0.67 | 0.60 | 0.17 | 0.67 | 1.00 | 1.33 | 1.67 | 2.00 | 2.34 | 2.67 | 0.83 | 1/6 | 1 | 5 | 6 |
| 0.86 | 0.70 | 0.20 | 0.86 | 1.20 | 1.60 | 2.00 | 2.40 | 2.80 | 3.20 | 1.00 | 1/5 | 1 | 4 | 5 |
| 1.00 | 0.90 | 0.25 | 1.00 | 1.50 | 2.00 | 2.50 | 3.00 | 3.50 | 4.00 | 1.25 | 1/4 | 1 | 3 | 4 |
| 1.14 | 1.00 | 0.29 | 1.14 | 1.70 | 2.30 | 2.85 | 3.45 | 4.00 | 4.60 | 1.43 | 2/7 | 1 | 2½ | 3½ |
| 1.33 | 1.16 | 0.33 | 1.33 | 2.00 | 2.67 | 3.33 | 4.00 | 4.66 | 5.33 | 1.66 | 1/3 | 1 | 2 | 3 |
| 1.60 | 1.40 | 0.40 | 1.60 | 2.40 | 3.20 | 4.00 | 4.80 | 5.60 | 6.40 | 2.00 | 2/5 | 2 | 3 | 5 |
| 2.00 | 1.75 | 0.50 | 2.00 | 3.00 | 4.00 | 5.00 | 6.00 | 7.00 | 8.00 | 2.50 | 1/2 | 1 | 1 | 2 |
| 2.50 | 2.20 | 0.62 | 2.50 | 3.75 | 5.00 | 6.25 | 7.50 | 8.75 | 10.00 | 3.12 | 5/8 | 5 | 3 | 8 |
| 2.67 | 2.33 | 0.67 | 2.67 | 4.00 | 5.33 | 6.67 | 8.00 | 9.33 | 10.67 | 3.33 | 2/3 | 2 | 1 | 3 |
| 3.00 | 2.62 | 0.75 | 3.00 | 4.50 | 6.00 | 7.50 | 9.00 | 10.50 | 12.00 | 3.75 | 3/4 | 3 | 1 | 4 |
| 3.20 | 2.80 | 0.80 | 3.20 | 4.80 | 6.40 | 8.00 | 9.60 | 11.20 | 12.80 | 4.00 | 4/5 | 4 | 1 | 5 |

of diluent which must be used; (5) dip off the proper milk and dilute all or a part of it, depending on the quantity of the food to be made up; (6) the addition of 2½ fairly level tablespoonfuls of milk-sugar or 2 exactly level tablespoonfuls of granulated sugar for about every 20 ounces of the total mixture will give the proper percentages of sugar."

## THE ROTCH LABORATORY METHOD OF MODIFYING MILK

The world owes to T. M. Rotch, of Boston, a lasting debt of gratitude for the work he has done in outlining, establishing, and popularizing percentage feeding. In accomplishing this work he has used as his agents the Walker-Gordon Milk Laboratories. These laboratories have the following stock supplies:

1. A 32-per-cent. fat cream mixture.
2. Separated milk which is almost fat-free.
3. A 20-per-cent. solution of milk sugar. Other sugars such as maltose, sucrose, and dextrose may be prescribed.
4. Whey.
5. Cereal decoctions.
6. Lime water and other alkalies used in modifying milk.

From these supplies the physician may prescribe a milk mixture calling for specified percentages of fat, protein, carbohydrates, and alkalies, designate the amount in ounces for each feeding and the number of feedings in twenty-four hours. The whole supply to be delivered each morning in a neat, clean box with compartments for holding the individual bottles and each bottle containing the amount of the mixture prescribed for a single nursing. The nurse or mother has only to warm the milk mixture by placing the bottle in warm water and then, on taking out the cotton stopper and slipping a nipple over the mouth of the bottle, it is ready for the baby to take.

The milk laboratories under the Walker-Gordon management are as reliable as milk laboratories can be made. The stock materials from which the modified milk is compounded are as clean and wholesome as scientific methods can make them. And the compounding is done with such accuracy that the physician may rely upon getting the percentages of the various ingredients as he has ordered them.

The following is a typical laboratory prescription:

| | Per Cent. | Remarks |
|---|---|---|
| ℞ Fats | 3.00 | Number of feedings ..... 9 |
| Milk sugar | 6.00 | Amount at each feeding... 3 ounces |
| Protein total | 1.00 | Infant's age..... 1 month |
| " (whey) | 0.75 | Infant's weight..... 9 pounds |
| " (casein) | 0.25 | Alkalinity, lime water ..... 5 per cent. |
| | | Heat at ..... 150° F |

If the original formula prescribed for the baby does not agree with it, or does not meet its nutritional demands, any one or all of the various

ingredients may be changed at the will of the physician by simply writing a new prescription. The laboratory method of percentage feeding is the simplest and most accurate. The expense, however, of thirty to forty cents a day which it entails, and the location of these laboratories in only a few of our largest cities shut out the vast majority of infants from the advantages which this method offers.

## ADDITION TO DIET OF BOTTLE-FED INFANTS AND CHILDREN

**Foods Added in the First Twelve Months of Infancy.**—The exact time at which other food shall be added to the diet of the bottle-fed infant depends altogether upon the digestive capacity of the individual, and what is here said applies to the average normal infant. Of course delicate infants of the same age will have to be placed upon a diet suitable to an infant several months younger; and strong, sturdy infants of unusual physical development may be able to take food which in the average is suitable for children several months older.

Orange juice is one of the most valuable of infant foods and is almost indispensable when the infant is fed upon sterilized foods. Under these conditions it prevents scurvy and overcomes constipation. It may be added to the diet at the eighth month. The juice of half an orange is to be given daily between feedings. At the end of the first year the quantity may be increased to a whole orange.

Meat juice is also a valuable food which may be begun as early as the eighth or ninth month, one ounce twice a day with a bottle feeding. Both the orange and the meat juice remain as staple articles of the infant's and child's diet for two or three years.

**Foods Added to the Diet from the Twelfth to the Eighteenth Month.**—Cereals, covered with clean milk and sugar or salt, may be begun at the end of the first year. A tablespoonful of well-cooked cereal, followed by six ounces of milk, making one of the meals. The following cereals are recommended: Oatmeal, cream of wheat, wheatena, and rice. In the beginning not more than one cereal feeding should be given in a day. Broths made from beef, lamb, or chicken and slightly thickened with one of the cereals may next be added to the diet; not more than six ounces of broth should be given and this should be followed by a six-ounce bottle, making a meal. Fresh, soft-boiled eggs are perhaps the most valuable addition to the infant's diet at this time. They are easily digested and in the beginning it may be wise to alternate with the cereals, giving an egg every second or every third day. The white of the egg mixed with eight ounces of water is a food that may be used even during the first year of life. Bread, toast, rusk, and crackers, softened with milk, may be given as a part of one of the meals at this time.

The normal infant from fifteen to eighteen months of age should have five feedings daily, and four of these feedings should be supplemented as

follows: A cereal with the first meal, broth with the second, egg with the third, bread or toast with the fourth, and following each of these a six-ounce bottle of whole milk. The orange juice still remains a part of the diet.

**Foods Added from the Eighteenth to the Twenty-fourth Month.**—Prune juice or apple sauce may be added to the six-o'clock meal at this time. Potato, baked, or boiled and mashed, may now alternate with the cereals. Beef balls made by broiling scraped beef may alternate with the egg and later take the place of the broth. During this period the ten-o'clock feeding is discontinued and the child has but four feedings a day.

**Foods Added during the Third Year.**—Fresh vegetables such as asparagus tips, peas, string beans, stewed celery, and spinach are now a part of the child's diet. One of these may be given each day. Desserts such as rice pudding, bread pudding, baked custard, junket, and ice cream made from clean milk may be gradually added to the child's diet; ice cream, however, should not be given more than once or twice a week and then only toward the end of the third year. Raw fruits, such as peaches, apples, pears, and grapes, may now occasionally take the place of apple sauce or orange juice. Meats, such as lamb chops, chicken, fish, or beef, may be minced and moistened with beef juice or broth and given once a day. Breads of various kinds, such as corn bread and dry cold biscuits, are now wholesome articles of food. By the end of the third year the child should have but three meals a day.

**From the Fourth to the Sixth Year.**—From the fourth to the sixth year the following foods should be excluded from the child's diet. Tea, coffee and alcoholic drinks, pastry, nuts and sweets, except very simple cakes and an occasional piece of good candy, pork, preserved meats, raw vegetables, salads, griddle cakes, and fried food of all kinds.

From the above diet list it will be easy to prescribe a diet suitable to the age of the child. But throughout all of this time it is most important that milk should remain the basis of the child's diet and care should be taken that it should not be overfed. There is little or no danger in underfeeding the normal child.

# SECTION IV

## *DISEASES OF DIGESTIVE SYSTEM*

## CHAPTER XV

### DENTITION

### THE TEMPORARY TEETH

**Eruption of the Temporary Teeth.**—The mouth of the infant for the first six or eight months of life contains no teeth. The absence of teeth at this time serves a wise purpose in that the infant can better perform the act of sucking. The formation of the temporary teeth begins in early fetal life and continues until at birth they are inclosed in membranous tooth sacs, more or less deeply imbedded in the alveolar processes of the jaws, covered only by the submucous connective tissue and the mucous membrane. The temporary teeth are twenty in number, and at birth the dental sacs which hold them rest upon the sacs of the permanent teeth. The eruption of the temporary teeth through the gums begins between the sixth and the eighth month and is usually not completed until in the early months of the third year of life. These teeth usually make their appearance in crops or groups of two or four, followed by a six or eight weeks' interval of rest. The eruption of the temporary teeth, on the whole, follows a definite order which may vary even in well infants, but these variations are greatly exaggerated in rachitic, syphilitic, and otherwise malnourished infants. On the whole, the teeth come through earlier and conform more closely to the regular order of eruption in the breast-fed than in the artificially nourished infant. The following table shows the usual order and the average time of eruption of the different groups of teeth. These variations are within normal physiological limits:

| | |
|---|---|
| Two lower central incisors | 6 to 8 months |
| Two upper central incisors | 8 to 12 months |
| Two upper lateral incisors | 9 to 12 months |
| Two lower lateral incisors | 12 to 15 months |
| Four anterior molars | 13 to 16 months |
| Four canines | 18 to 22 months |
| Four posterior molars | 22 to 30 months |

When one year of age an infant should have six teeth; when one and one-half years of age, ten or twelve teeth; when two years of age, sixteen

teeth; when two and one-half years of age, twenty teeth. In rare instances infants may be born with teeth; when this occurs it is almost always the lower central incisors that are present. This congenital anomaly is of little pathological importance. In many instances these teeth are set so loosely in the gums that they act as an irritant and interfere with nursing; when this is the case they should be removed. When, however, they are firmly set in their alveolar processes they should be let alone; their presence causes neither the infant nor the mother any inconvenience and their removal deprives the infant of just so many temporary teeth.

**Delayed Dentition.**—Rickets and other forms of malnutrition are the common causes of delayed dentition, but heredity may also be a factor. If no teeth have appeared by the end of the first year of life, rickets should be suspected and other symptoms of this disease sought for. The same causes which delay dentition predispose to irregularities in the time of eruption of the various groups; for example, the upper incisors may appear before the lower and the canines before the molars. In malnourished children the teeth not only come in late and irregularly, but they are poorly developed, imperfectly formed, and decay early.

**Dentition as a Pathological Factor.**—At one time in the history of medicine almost all the ills of infancy and early childhood which occurred during teething were attributed in a greater or less degree to dentition. At that time it was believed to be the all-important cause of gastrointestinal disturbances and of functional nervous disorders. Diarrhea, enteritis, sleeplessness, general nervous irritability, convulsions, running ears, and even diseases of the respiratory passages were attributed to dentition. In recent years, as the causes of diarrheal, nervous, and other diseases in infancy have been more carefully worked out, dentition has gradually lost its importance as a pathological factor. The teachings of Forchheimer played no little part in this reaction. He strongly maintained that dentition rarely played any rôle either in producing or aggravating pathological processes, and taught that "teething produces teeth and nothing more." While Forchheimer recognized that certain slight and evanescent symptoms might be due to teething, he held that the safety and welfare of the infant, during this period, largely depended upon the clinician disregarding dentition as a pathological factor and searching for other remedial causes of the infant's illness. However, it is important that we should not altogether forget that teething is not infrequently accompanied by pain and by certain symptoms on the part of the nervous system and gastrointestinal tract. In a perfectly normal breast-fed infant a tooth may come through without producing any symptoms whatever; the first evidence of its eruption may be the finding of its tiny point, which has pierced the gum over-night, but this is not usually the case even in normal infants. The most common symptoms due to teething are swelling, redness and tenderness of the gums, increased flow of saliva, sleeplessness, marked restlessness, increased nervous irritability, exaggerated reflex excitability, elevation of temperature, refusal to take food, regurgitation of food, vomiting, intestinal indigestion,

and slight looseness of the bowels. In rachitic and malnourished infants these symptoms are so much more marked that one might almost say that their severity is largely dependent upon the degree of malnutrition from which the infant is suffering.

**Care of the Teeth.**—It is rarely if ever necessary to lance the gum to relieve the symptoms which are believed to be associated with difficult dentition. The majority of pediatricians never find it necessary to make this operation. It is important to take proper care of the temporary teeth, since they serve the purpose of preserving the shape of the jaw and making second dentition more normal and less difficult; they are to be kept clean by washing the mouth once or twice a day with lukewarm water, or with a weak boracic solution. Particles of food materials must not be allowed to collect between the teeth or at their roots, and thus furnish a culture field for pathogenic microorganisms.

## THE PERMANENT TEETH

Permanent teeth are thirty-two in number. The first molars, which appear earliest, penetrate the gum about the sixth or seventh year. Forchheimer's table, which follows, gives the time of the appearance of the second set of teeth.

| | |
|---|---|
| First molars | 6 years |
| Incisors | 7 to 8 years |
| Bicuspids | 9 to 10 years |
| Canines | 12 to 14 years |
| Second molars | 12 to 15 years |
| Third molars | 17 to 25 years |

Malnutrition of a pronounced type may delay and interfere with second dentition just as it does with first dentition. In congenital syphilis the second teeth are poorly formed, decay early and in some instances the upper central incisors show a characteristic deformity known as Hutchinson's teeth. This condition is described under Congenital Syphilis. In neurotic malnourished children the eruption of the second teeth may act as a reflex factor in the production of nervous symptoms and digestive disturbances somewhat less severe than the symptom group produced by the first dentition.

# CHAPTER XVI

## STOMATITIS

## STOMATITIS CATARRHALIS

**Symptomatology.**—Stomatitis catarrhalis is a simple inflammation of the mucous membrane of the mouth. It usually begins on the gums or tongue and spreads to involve the entire mucous membrane of the mouth. It is characterized by redness and swelling of this membrane, and by an

increasing salivary and mucous secretion. When the disease is well established the mucous membrane is intensely congested and slight hemorrhages may occur. The gums are usually much swollen, and this may extend to the lips, causing a decided thickening. The pain and irritation cause the infant to be fretful, sleepless, and to refuse food. The act of sucking apparently causes pain. The salivary glands are excited to increased activity, so that the saliva flows out of the mouth, wetting the face and clothing. Forchheimer calls attention to the enlargement of the muciparous follicles which appear as small, round prominences on the red mucous membrane, and to the fact that in older infants the swollen tongue and cheeks show the indentations of the teeth. The temperature is normal or slightly elevated. The lymph nodes are not enlarged. The infant's general nutrition may suffer slightly because of the lack of food and general nervous irritability. When constitutional symptoms are marked the catarrhal stomatitis is then a symptom of some acute toxic condition.

**Etiology.**—This condition is most common during the first year of life. It is produced by some mechanical, chemical, toxic or thermal injury to the mucous membrane (Forchheimer). The introduction of foreign bodies, carelessness or roughness in washing the mouth, strong acids or alkalies, dirt, decomposing food, pacifiers, and hot and cold food are among the common exciting causes. Catarrhal stomatitis is usually present in most of the acute infectious diseases.

**Treatment.**—The disease runs a benign course and terminates in recovery within five or six days, provided the mouth is kept clean and the exciting cause is removed. The mouth may be washed with a 2 or 3 per cent. solution of boracic acid or with a mild alkaline antiseptic. A laxative should be given, preferably castor oil, and the milk, if the infant be artificially fed, should be diluted with barley water; cool food is more readily taken. It is, as a rule, bad practice to attempt to force food in these cases; in some instances, however, the modified milk mixture will be taken readily with a spoon even after the infant has refused to nurse. Weak solutions of some astringent, such as a one-half per cent. solution of nitrate of silver, is recommended in those cases where for any reason the disease is prolonged beyond a week.

## STOMATITIS APHTHOSA

**Etiology.**—The causes of this condition are not definitely known. It has been suggested that it is brought about by intestinal or other toxins; that it is of neurotic origin; that it is a local infection due to pathogenic microorganisms and associated with uncleanliness and fermenting food material in the mouth. It is sometimes associated with the acute infectious diseases and with severe disturbances of the gastrointestinal canal.

**Symptomatology.**—This condition has been described under the name herpetic stomatitis, maculofibrinous stomatitis, vesicular stomatitis and follicular stomatitis. It is characterized by the appearance of superficial ulcers, scattered widely over the soft palate, hard palate, gums, tongue, and

the inner surface of the lips and cheek. These ulcers appear as yellowish-white spots, covered with a fibroplastic exudate and surrounded by an area of congested, swollen and reddened mucous membrane. There may be only a few of these ulcers present, or the whole mucous membrane of the mouth may be dotted with them. These small, round, yellowish-white ulcers, from one-eighth to one-fourth of an inch in diameter, present a very characteristic appearance. If near together they may coalesce to form larger, irregular ulcers. The catarrhal stomatitis, which is always associated with this condition, is more intense in the immediate vicinity of the ulcers. There is great increase in the salivary flow, and drooling is always present. The pain is very intense and is greatly aggravated by any irritating material coming in contact with the ulcers. For this reason infants suffering from this condition may abstain almost absolutely from food for two or three days at a time, and a paroxysm of crying and nervous irritability may be provoked by any attempt at feeding. This condition occurs most commonly during the second year of life, so that children suffering from it can usually be induced to take water or some non-irritating food such as milk or barley water from a spoon when they absolutely refuse to take it from a nursing bottle. Not infrequently gastrointestinal disturbances are associated with this form of stomatitis.

**Prognosis.**—This disease runs a benign course and terminates in recovery within a week or ten days.

**Treatment.**—This should be begun with a dose of castor oil, to be followed by a very bland diet. Irritating food, such as broths and meat juice which contain salt, cause pain and are refused. Cool water and cow's milk diluted with barley water should be given to the child to drink or should be fed with a spoon. The mouth should be washed with mild alkaline non-irritating antiseptics; a weak solution of boracic acid may be used for this purpose.

## STOMATITIS MYCOSA

(*Sprue; Thrush*)

**Etiology.**—Thrush is produced by a specific fungus which, under the microscope, presents the appearance of fine, tangled, jointed filaments. These slender threads are composed of rods with spores at their ends, and scattered through the tangled threads isolated spores and epithelial cells are seen. These fungi, as Forchheimer has taught, penetrate beneath the epithelial layers, lifting and separating them; in this way the disease spreads along the surface and into the deeper layers of the mucous membrane and in rare instances invades the underlying tissues and distant organs. It most commonly attacks the mucous membrane of the cheeks, hard palate and tongue; other portions of the mouth are not uncommonly involved, and much more rarely the pharynx, stomach and intestines are attacked by this fungus. It occurs most commonly during the first three months of life among infants whose surroundings are dirty and unhygienic

and who are improperly fed and neglected. Catarrhal stomatitis usually precedes thrush and prepares the mucous membrane for inoculation with the fungus of this disease. All the exciting factors of catarrhal stomatitis become, therefore, the predisposing factors of thrush. This disease is very much more common in malnourished, marasmic infants and is therefore more prevalent in hospital and dispensary practice. The contagion is very widespread; it may be found on the buccal mucous membranes of healthy infants. This impresses the fact that susceptibility to this disease is an important etiological factor. Normal mucous membranes are capable of resisting it, but diseased and injured mucous membranes furnish a favorable soil for its growth. General lack of resistance, associated with profound malnutrition, is also an important factor, not only in starting the growth, but in facilitating its spread.

**Symptomatology.**—This disease is readily recognized by the appearance on the tongue, cheeks or other portions of the mouth of small, white patches which resemble coagulated milk. These white masses seem to be loosely attached, but on attempting to remove them they are found to cling closely and to be imbedded in the mucous membrane; their removal leaves raw and bleeding surfaces; by this fact they are easily differentiated from milk curds. As they increase in size they coalesce and form larger irregular patches, and in aggravated cases may cover a large portion of the buccal mucous membrane. A slight catarrhal stomatitis is usually present. The secretions of the mouth take on an acid reaction due to the fermentation produced by this fungus. There may be more or less difficulty in swallowing and pain on taking food, which causes the infant to refuse nourishment. This may become a serious feature of the disease when it occurs as a complication of severe malnutrition, and may in the marasmic infant hasten or cause a fatal termination. Uncomplicated thrush, however, is not a serious disease; it runs its course and terminates in recovery within a week. When associated with severe constitutional disorders, however, the disease may persist an indefinite length of time, and recurrences in these cases are not uncommon. In rare instances tonsillar or pharyngeal thrush may somewhat resemble diphtheria, but the age of the infant, the absence of constitutional symptoms, and, finally, a microscopical examination which reveals the thrush fungus will readily make the differential diagnosis.

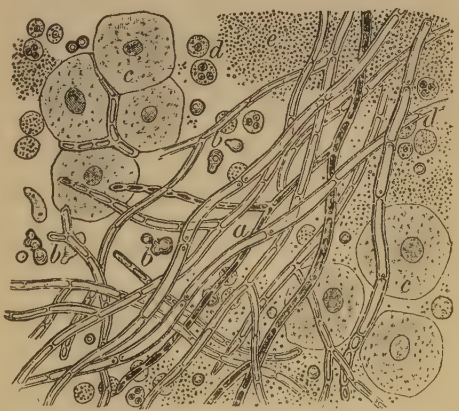



FIG. 22.—THRUSH FUNGUS (HIGHLY MAGNIFIED). *a*, mycelium; *b*, spores; *c*, epithelial cells from the mouth; *d*, leukocytes; *e*, detritus. (V. Jaksch.)

**Treatment.**—The prophylactic treatment of thrush has greatly dimin-

ished its prevalence in recent years. If the infant is properly cared for and is given clean food through clean nipples and is protected in other ways from the introduction of filth and dirt into its mouth, thrush will rarely occur. The tendency at the present time is to overdo in the matter of mouth-washing in the healthy infant. Whatever mouth-washing is done during the first few months of life should not only be done with sterile washes and soft sterile cloths, but it should be done gently so as not to injure the buccal mucous membrane. Pacifiers, dirty toys and other unclean things should be kept out of the infant's mouth, and above all it should be fed carefully along the lines outlined under Infant Feeding.

In the treatment of thrush a dose of castor oil should be given, and thereafter the child should be carefully fed as above indicated. Gastrointestinal disorders and the underlying malnutrition, if such exist, should be given appropriate treatment. If the infant is breast-fed, the mother's nipple, before and after nursing, should be carefully cleansed. If it refuses food it may sometimes be necessary to use gavage; in most instances, however, the infant may be fed with a spoon or with a medicine dropper.

The local treatment of this condition is simple. The patches should be gently brushed or wiped with cotton or a soft cloth saturated with a mild alkaline antiseptic; a 2 per cent. solution of boric acid and bicarbonate of soda serves this purpose well. In order to remove the patches quickly this brushing should be done three or four times a day; care should be taken, however, not to injure the mucous membrane by forcibly tearing the patch away. Stronger applications, such as a 1 per cent. nitrate of silver solution, may be gently applied once a day in those cases where the thrush does not yield readily to the boric acid treatment. Following the use of boric acid or nitrate of silver the mouth should be washed with sterile water.

## STOMATITIS ULCEROSA

**Etiology.**—The specific cause of this disease is not definitely known, but the distinct clinical picture which it presents indicates that it is due to some specific microorganism. Bernheim and Pospischill, in a series of cases, isolated a bacillus probably identical with that of Vincent, which was always associated with a spirochete, and they apparently demonstrated that ulcerative stomatitis could be produced by these microorganisms. The fact, however, that these microorganisms are also associated with mercurial stomatitis, with various forms of gangrene and suppurative diseases about the mouth, together with the fact that in ulcerative stomatitis, streptococci, staphylococci, and other pus formers play an important rôle in the destructive process, indicates that the etiological relationship of the Vincent bacillus to stomatitis ulcerosa has not been definitely proven. This disease occurs most commonly in malnourished children having decayed or diseased teeth, which irritate and mutilate the gums and furnish a nest for decomposing food material. Improper food, uncleanliness of the food utensils and everything that facilitates the carrying of dirt and micro-

organisms into the child's mouth may be etiological factors. This disease is much more common in hospital and dispensary practice.

Ulcerative stomatitis also occurs as a symptom of scurvy and of mercurial and other metallic poisonings.

**Symptomatology.**—The offensive breath and profuse salivation are the symptoms which commonly call attention to this disease, and an examination of the mouth reveals in the early stage an intense redness and swelling of the gums, usually along the incisor teeth. The swelling and redness become more marked, the gum separates slightly from the tooth and a yellowish ulcer appears on its edge. As the disease progresses the ulceration spreads and extends to the buccal mucous membrane which is opposed to the ulcer. The extent of the ulceration on the two opposed mucous membranes corresponds very closely. As the ulceration proceeds the gum becomes more and more separated from the teeth, which may be loosened and sometimes may be lost. The ulcerated mucous membrane is very tender, bleeds on the slightest touch, and causes great discomfort, especially when food is taken. The offensive breath becomes more fetid, the saliva pours from the mouth, and the large ulcer, which has resulted in considerable loss of tissue, both on the gum and the opposed mucous membrane of the lips or cheeks, is covered with a yellowish, purulent exudate. In rare instances the alveolar processes may be involved and necrosis of bone may occur. The anterior cervical lymphatic glands are swollen and tender. The tongue is covered with a thick, brownish coat. There are no constitutional symptoms produced by this disease; when fever and other general symptoms are present they are due to a coexisting or complicating affection.

The course of the disease is usually benign and recovery may be expected within a week or ten days. When, however, this condition occurs as a complication of severe malnutrition it does not yield so readily to treatment.

**Treatment.**—The preventive treatment is the same as that given for thrush. The local treatment consists in the careful washing of the ulcer with some mild alkaline antiseptic: a 2 per cent. boracic acid and bicarbonate of soda solution may be used for this purpose. The ulcer should not be irritated by the application of strong astringents or by brushing it roughly with cloth or cotton for the purpose of applying a cleansing solution; if it is carefully exposed, gentle irrigation is all that is necessary for purposes of cleanliness.

Chlorate of potash is the all-important remedy in the treatment of this disease; it is believed by Forchheimer and others to act specifically in its cure. One or two grains of chlorate of potash, depending upon the age of the child, is to be given, well diluted, every hour or two during the waking period for two or three days; thereafter, if necessary, it should be given at longer intervals. A solution of chlorate of potash should also be used at intervals during the day for irrigating the ulcer. The chlorate of potash taken internally is largely excreted through the saliva, and in this way a more or less constant application of this drug to the diseased parts is pro-

duced. Weak solutions of alum and nitrate of silver are also very generally recommended, especially in those cases that do not yield readily to treatment. In my experience these astringents are rarely, if ever, necessary. An important part of the treatment consists in inducing the child to take proper food, such as milk and cereal mixtures.

## STOMATITIS GANGRENOSA

*(Noma; Cancrum Oris)*

**Etiology.**—Noma is a severe infection most commonly beginning in the mucous membrane of the mouth and resulting in more or less extensive gangrene of the soft parts of the face; its definite clinical history indicates that it is due to some specific cause. Babes and Zambolovici isolated a bacillus which they believe to be etiologically related to this disease, and the bacillus and spirillum of Vincent together with the ordinary pyogenic

FIG. 23.—STOMATITIS GANGRENOSA, BEFORE PERFORATION.

FIG. 24.—STOMATITIS GANGRENOSA, AFTER PERFORATION.

organisms are associated with its destructive processes. Noma occurs most commonly between the second and the sixth year of life. It attacks children whose vital powers have been greatly reduced by serious illness. It may occur as a sequel of ulcerative stomatitis and is commonly seen as a complication of measles and, more rarely, of diphtheria, typhoid fever, scarlet fever and whooping-cough. Holt saw "five cases in a single ward, all beginning in the auditory canal, which were apparently produced by the use of the same syringe to clean the ears without proper disinfection. All of these children were suffering from whooping-cough at the time."

**Symptomatology.**—A putrid odor may lead to the examination of the

mouth, and the diagnosis is made by the characteristic appearances there seen. On the inside of the cheek a dark, necrotic ulcer, surrounded by an infiltrated and swollen area, may commonly be seen and felt, and the outer surface of the cheek is infiltrated, producing a hard lump, over which the skin may be pale and not at all sensitive to touch. As the infiltration spreads a well-defined dark or black, gangrenous patch may be seen on the mucous membrane. The necrosis slowly spreads, the gangrenous process extends through the cheek, involving the skin. The line of demarcation between the dead and the live tissue is now well marked. Perforation of the cheek results from the dislodgment of the gangrenous patch, and through the opening the teeth or mucous membranes of the gums may be seen. Very commonly the gums are affected, and the bones beneath may become necrotic and the teeth may come out. There is little or no pain in this disease and hemorrhages are very unusual. Strangely enough the gangrenous process is almost always confined to one side of the face; rarely both sides may be affected. As the disease progresses the fetor increases and the disagreeable odor is almost unbearable. In the beginning there is little to call attention to the serious character of the disease; the child may be playful, sit up in bed and take nourishment, but as the disease progresses a septic temperature becomes more marked. The fever may reach 104° or 105° F. The pulse becomes weak, prostration grows apace, and the child finally dies from exhaustion, or from some complication such as bronchopneumonia.

**Prognosis.**—The prognosis is very grave. The great majority of these cases die. Fifteen or twenty per cent. of them recover after prolonged illness, and the resulting deformity is great.

**Treatment.**—There is little in the way of medical treatment beyond careful feeding and the use of whiskey, brandy and heart tonics to keep up the general strength of the child; the gangrenous parts should be kept as clean as possible by antiseptic washes. In a small percentage of the cases nature effects a cure. Diphtheria antitoxin has been used with some success in cases due to the Klebs-Löffler bacillus. In gangrene, following tonsillitis, antistreptococcic serum may do good.

Noma should be classified as a surgical disease, and as soon as the diagnosis has been accurately established the case should be referred to a surgeon for treatment. Excision of the gangrenous part should be thorough; the tissues should be removed well beyond the gangrenous line and all diseased bone taken out; the surgical wound thus made should be cauterized and thereafter dressed according to approved surgical methods. Under this treatment the chances for recovery are improved, but at best the condition is a desperate one and desperate chances should be taken to save life.

## CHAPTER XVII

### OTHER DISEASES OF THE MOUTH AND DISEASES OF THE ESOPHAGUS

### BEDNAR'S APHTHÆ

This is a condition of little pathological importance. It consists of two small, rounded, grayish-white ulcers about the size of a pea, symmetrically located on the hamular processes of the palate bone, at an equal distance from the palatine ridge. They produce no constitutional symptoms, but are slightly painful to the touch and may therefore interfere with the taking of food. They are believed to be produced by some injury to the mucous membrane, which is especially prominent and delicate over the hamular processes. The exciting causes are too vigorous mouth-washing and the prolonged sucking of rubber nipples and pacifiers. This condition occurs only during the first weeks of life.

**Treatment.**—The condition is a benign one and requires no treatment other than the irrigation of the mouth with sterile water or a 2 per cent. solution of boric acid. The ulcers are not to be rubbed or cleansed with gauze or cotton; they should be let alone. Even the untreated cases terminate in spontaneous recovery. To hasten the cure it has been recommended that the ulcers be lightly touched with a 1 per cent. solution of nitrate of silver once a day.

### PERLÈCHE

Perlèche is an ulcerative process, probably nonspecific in character, which confines itself to the angles of the mouth. It occurs throughout childhood, but is perhaps most common about the second or third year of life. It is commonly seen in children suffering from glandular tuberculosis and other forms of general malnutrition. Lack of cleanliness in the care of food utensils and unhygienic surroundings predispose to this infection. It may be transferred to other members of the family by direct contact, as in kissing, or by indirect means, such as the use of common food utensils and toilet articles without proper sterilization. One corner of the mouth is nearly always infected from the other by the tongue transferring the contagion. This accounts for the bilateral character of this disease.

**Symptomatology.**—The corners of the mouth are the sites of ulcers which first make their appearance in the form of slight fissures; later the ulceration extends and is covered by a sticky exudate and the surrounding parts are swollen and indurated. Pain and slight bleeding are produced by stretching the corners of the mouth and forcible removal of the scab leaves a raw and bleeding surface. From these ulcers there radiate from the corners of the mouth well-marked fissures, giving to this condition a characteristic appearance, which cannot be mistaken for any other form

of ulceration except the syphilitic ulcer which occurs at this point. The differentiation between these two conditions, however, may be made by the existence or absence of other syphilitic symptoms.

**Treatment.**—In the great majority of cases even the untreated cases terminate in spontaneous recovery; in a few instances, however, the disease may continue for months. The scabs covering the ulcers should be softened by the application of a 1 per cent. boracic acid ointment; after a number of days they may be removed without producing much irritation. The surfaces thus exposed should be painted two or three times a day with a 2 per cent. nitrate of silver solution. Following the application of the silver the ulcers should be washed and some such sedative ointment as the following should be applied: Bismuth subnitrate, ℥i, acid salicyl., grs. xx, lanolin, q. s. ad ℥i.

## ELONGATED UVULA

An elongated uvula may, by irritating the base of the tongue and the pharynx, produce a most persistent and irritating cough, which may be greatly aggravated when the child is lying down. As a result of constant coughing the child loses sleep and becomes more or less nervous and hysterical, and this nervousness in turn may increase the paroxysms of coughing. When this symptom group occurs in children having a normal temperature and with no physical signs in the throat or lungs to account for it the uvula should be inspected, and if it be found elongated to such an extent that it can readily come in contact with the pharynx and tongue, it should be amputated. Clipping off the uvula is a simple operation which effectually terminates the existing attack and prevents recurrences.

Astringent gargles or astringents applied in other ways to the uvula are effective in relieving the attack. A twenty per cent. solution of alum or 5 to 10 per cent. solution of tannic acid, when applied to the uvula, usually causes it to contract and relieves the irritating cough. A good-sized dose of bromid of potash should be given at the same time.

## GEOGRAPHICAL TONGUE

(*Ringworm of the Tongue; Desquamative Glossitis; Wandering Rash of the Tongue*)

**Symptomatology.**—One or the other of these names is used to describe a condition of the tongue which has little or no pathological significance, but the very remarkable change which it produces in the surface of the tongue always attracts attention and calls for explanation. There appears usually on the dorsum a grayish-white patch distinctly outlined by the surrounding pink mucous membrane; as this increases in size it takes on a characteristic appearance; in the center it becomes more or less denuded of the superficial epithelial layers, which gives it a reddish color. As these red patches increase in number they may coalesce and give to the tongue a geographical appearance. If the scrapings from these elevated borders be

placed under the microscope, in addition to the epithelium and detritus thus obtained there will be found cocci, sarcinæ and other microorganisms, none of which have as yet been definitely associated with the etiology of this condition. It occurs most commonly from the first to the fifth year of life, but it may be seen at any time during infancy and childhood.

**Treatment.**—This condition is of no diagnostic or pathologic importance. It may occur in perfectly normal children and its presence furnishes no clue to the existence of any constitutional or local disorder. It requires no treatment, although painting the tongue with 5 or 10 per cent. solutions of nitrate of silver and afterward thoroughly irrigating the mouth with a mild alkaline antiseptic have been recommended. This condition may persist for months or years and then disappear, or it may continue throughout life.

## TONGUE-TIE

A short frenum which binds the tongue to the floor of the mouth is the cause of this deformity. It interferes with sucking and articulation and prevents the protrusion of the tongue beyond the gums.

The diagnosis of tongue-tie in backward children is frequently made, when it does not exist, to account for their defects in articulation. The treatment consists in cutting the frenum and separating the tissues far enough back to liberate the tongue.

## HARE-LIP

In the formation of the upper lip a central process unites with two lateral processes just beneath the nostrils. When this union fails to occur the lip remains fissured or slit on one or both sides, producing a single or double hare-lip. The deformity may vary from a slight indentation to a fissure completely separating the lip and extending into the nostril. When this deformity occurs on both sides it is much more difficult to overcome by surgical interference.

Hare-lip greatly interferes with the feeding of the infant and sometimes makes nursing impossible. It is important in all cases to encourage nursing and supplement the feeding by pumping out the mother's milk and feeding it with a dropper. As soon as the nutritional processes of the body have been well started a surgical operation for the relief of this condition is advisable. This may usually be done about the fourth week of life.

## CLEFT-PALATE

Cleft-palate is commonly associated with hare-lip and not infrequently with other congenital deformities. Heredity is the most common etiological factor; not infrequently more than one case occurs in the same family. This deformity is said to occur more commonly in boys; I observed, however, one family in which there were four girls all born with cleft-palates and hare-lips, and three boys entirely free from these deformities. The

fissures may involve only the soft palate or both the hard and soft palate. It results from failure of the palatal arches to unite. Cleft-palate, especially when associated with hare-lip, is a serious deformity and not infrequently interferes to such an extent with the taking of food that the infant dies from marasmus. This deformity makes it difficult to keep the infant's mouth clean and thereby predisposes to thrush.

The early treatment of these cases consists in devising ways and means by which the infant may be fed with breast milk. By pumping the breasts of a wet nurse milk may be secured, which may be fed to the baby by a spoon or dropper. The greatest care should be exercised to keep the infant's mouth clean by gently washing it with warm water. If the operation for hare-lip is successfully made during the second month, there will be less difficulty in feeding and the operation for cleft-palate may be deferred until the end of the second year. As the treatment, however, is essentially surgical, both the time and the nature of the operation may be left to the judgment of the surgeon.

## ESOPHAGITIS

Esophagitis is an inflammation of the esophagus commonly due to the swallowing of caustic alkalies or mineral acids or to the extension of some inflammation from the pharynx.

**Symptomatology.**—In caustic esophagitis the stomach is usually involved; there are great pain, restlessness, crying, and difficulty in swallowing; any attempt at taking food or water aggravates these symptoms. Nausea and vomiting are common and the vomited matter may contain blood. An examination of the mouth and throat shows that these mucous membranes are swollen and inflamed. The severity of the symptom group depends upon the quantity and concentration of the caustic fluid swallowed. In severe cases the injury to the stomach may cause a rapidly fatal termination. In the milder cases of esophagitis the above symptom group is less severe and may gradually subside; convalescence resulting either in complete recovery or in stricture of the esophagus.

**Treatment.**—This consists in relieving the pain when necessary by hypodermic injections of morphin and in the giving of nutrient and saline enemata until the child is able to swallow milk and water. Later surgical intervention may be necessary to relieve the esophageal stricture.

## PERIESOPHAGEAL ABSCESS

Periesophageal abscess is usually due to suppuration of lymph nodes, disease of the spine or to foreign bodies; tuberculosis is the most common cause.

The symptoms depend largely upon the location of the abscess and are usually those of esophageal stenosis. When located high up, the trachea, larynx and recurrent laryngeal nerve may be involved. In these cases

there may be dyspnea, aphonia and violent attacks of coughing. The abscess may cause death by opening into the esophagus or trachea.

The prognosis is bad; spontaneous rupture may rarely result in recovery. Surgical intervention is advisable in selected cases.

## BRANCHIAL CYSTS

Branchial cysts are cystic tumors having their origin in the faulty closure of the branchial clefts of fetal life. They are usually located in the anterior-lateral surfaces of the neck in close proximity with the great vessels. This rare form of cystic tumor yields readily to radical surgical treatment.

## CONGENITAL MALFORMATIONS OF THE ESOPHAGUS

Griffith notes the following forms of congenital malformations of the esophagus: "1. Total absence of the esophagus. 2. Partial or complete doubling of esophagus. 3. Tracheoesophageal fistula without other lesion of the esophagus. 4. Congenital stenosis. 5. Congenital dilatation. 6. Obliteration of the esophagus in only a portion of its extent unaccompanied by fistula. 7. Obliteration of a portion of the esophagus with tracheoesophageal (or bronchoesophageal) fistula.

"**Treatment.**—This is entirely discouraging. Cases of stenosis have recovered, but all instances of complete obstruction have died. The weakness of the child and its early age make operative interference a questionable procedure; yet gastrostomy offers the only hope. This operation was first done in the case of Steel, and has been performed since then in those of Hoffmann, Hoppich, Villemin, Kirmisson, and Dickie. The child should be kept on its side to allow the mucus to flow from the mouth. It may be fed through the gastric fistula. Should it recover an effort may later be made to repair the esophagus by a lateral operation in the neck. This indeed was attempted unsuccessfully in Hoffmann's case as a primary operation and gastrostomy resorted to later."

# CHAPTER XVIII

# DISEASES OF THE STOMACH

## ACUTE GASTRIC INDIGESTION

**Etiology.**—The causes of this condition, especially in the infant, may be grouped under two headings: first, Physiological Gastric Incompetency; second, Improper Food.

Physiological Gastric Incompetency.—Physiological gastric incompetency may be inherited or acquired; it is most pronounced during the hot summer months. In feeble, malnourished, neurotic infants there may be

such a predisposition to acute gastric indigestion that slight causes, such as rapid eating, coddling and exercise directly after meals, slight changes in the food formula, dentition, nervous excitement, or the swallowing of mucus in catarrhal conditions of the respiratory passages, may cause this trouble. It is important therefore in every case of acute gastric indigestion to consider not only the apparent exciting causes, but also the probable influence which the physiological gastric incompetency of the individual infant may play in the production of this symptom group.

Improper Food.—The most common exciting causes are to be found either in the quantity or in the quality of the food. It may occur in breast-fed babies from irregularities in nursing; that is to say, giving too much food at too short intervals. The breast milk itself, however, may cause indigestion by changes in its composition produced by nervous excitement on the part of the wet nurse, or by the character of her food, or by the unhygienic life she may be leading. Artificial food unsuited to the digestive capacity of the child, or properly prepared food given in too great quantities or at too short intervals, are the most common causes of acute gastric indigestion. Rapidly increasing the strength of the food formula, especially in fat and protein, or changing from one of the proprietary foods to a modified milk formula are common exciting causes. In older children the taking of improper food, or food beyond the digestive capacity of the child may produce very severe attacks of gastric indigestion. Candy, pastry, fruit, berries and vegetables given to infants and children whose age and digestive capacity wholly unfit them for the taking of these foods are common causes of gastric indigestion.

**Symptomatology.**—The attack is usually ushered in with colic, nausea, irritability, fretfulness, restlessness and a slight elevation of temperature. The appetite is lost, the tongue is coated and after a time vomiting occurs, the vomitus containing undigested food that has been perhaps retained in the stomach for many hours. The irritability of the stomach and vomiting may recur at intervals for a number of hours, and the taking of food may prolong and aggravate these symptoms. Following the vomiting the pain, fever and nervous symptoms gradually subside. During the next few days the stomach is irritable and vomiting is easily provoked, and more or less intestinal irritation with diarrhea is present. In young, delicate and malnourished infants all of these symptoms may be greatly exaggerated. The fever may run as high as 104° or 105° F. and great prostration, extreme pallor, great nervous irritability and even convulsions may occur. But in these severe cases, as in the milder ones, the emptying of the stomach by vomiting and the unloading of the intestinal canal by a cathartic quickly cause a subsidence of all the acute symptoms, leaving the child weak, and suffering from a gastric irritability from which it slowly convalesces under careful treatment. In older children the fever is, as a rule, absent and the nervous symptoms are much less marked, but gastric colic or severe gastralgia is a much more common symptom than it is in the infant.

**Prognosis.**—The prognosis is good and there is rarely any danger from

gastric indigestion except in young and very delicate infants, and then the danger is commonly due to convulsions. If the stomach is emptied and food is stopped the infant should be convalescent within three or four days.

**Treatment.**—If vomiting has not occurred, and even if it has and there is reason to believe that the stomach has not fully emptied itself, it is advisable to wash out the stomach with a lukewarm physiological salt solution; this procedure often arrests the nausea and vomiting. Small doses of calomel should be given, a tenth of a grain every half hour until one grain is taken, and all food should be stopped and only sufficient water given to administer the calomel. Rest for the stomach and quiet for the infant during the next few hours are necessary. After three or four hours a dose of milk of magnesia should be given to assist the calomel in its action. Castor oil, which is likely to prolong the gastric irritability, should be abstained from. Commonly no other medication is needed in these cases, but if the gastric irritation is prolonged, a teaspoonful of simple chalk mixture or lime water may be given every two or three hours, or sodium bicarbonate or compound chalk powder in 2-grain doses may be given in a little water every two hours. After some hours, when the infant demands food, it may be given water to drink and later barley water; if the infant be very young the barley water may be malted. Barley water, beef broth, beef juice and whiskey and water should constitute the nourishment for twenty-four or thirty-six hours, and then small quantities of fat-free cow's milk should be added to the barley water, and as the child convalesces it may gradually return to its original diet. In breast-fed babies the breast milk may be carefully resumed after the stomach has been rested for twelve or twenty-four hours.

In older children the treatment is somewhat different; the gastric pain must be relieved by hot applications to the stomach, spirits of chloroform and whiskey internally, and if the child has not vomited an emetic may be given, preferably the syrup of ipecac. Later, as the nausea, vomiting and pain subside, a saline cathartic should be given, the stomach should be rested and the child dieted according to its age and physical condition. The following prescription is of value during this period:

℞ Acidi hydrochlorici dil. ..... ʒ i
Pepsin puri ..... grs. xv
Glycerini ..... ʒ ii
Aquæ destillatæ ad ..... ℥ ii
Sig. Teaspoonful after eating for a child six years of age.

## ACUTE GASTRITIS

**Etiology and Pathology.**—Noncorrosive Form.—Acute gastritis is very commonly a sequel of acute gastric indigestion. In feeble, malnourished, neurotic children this sequence is most commonly noted. The prolonged heat of summer may, by reducing the vitality of the infant, predispose it to attacks of gastritis. Spoiled food, especially milk which has undergone bacterial contamination, is a potent factor. Gastritis is also

a common complication of the acute infections, especially influenza, measles and whooping-cough. Acute enteritis, whatever may be its causes, is very commonly complicated with acute gastritis, and an acute gastritis which does not yield promptly to treatment is almost always followed by more or less enteritis.

In the ordinary form of acute gastritis the stomach is found to contain a thick, tenacious mucus, closely adherent to the mucous membrane, and this mucus may be mixed with a dark granular substance which analysis proves to be blood. The mucous membrane presents the appearance of an acute catarrhal inflammation. It is hyperemic, swollen, thickened, is infiltrated with round cells, shows a superficial loss of epithelium and may be dotted with petechial hemorrhages. Small ulcers, similar to those which occur in gastroenteritis, may be present in the more severe cases; a pseudomembrane rarely occurs.

Corrosive Gastritis.—Corrosive gastritis differs radically in its etiology and clinical history from ordinary gastritis. This severe form of inflammation of the stomach may be excited by such caustic poisons as ammonia, carbolic acid, mineral acids, arsenic and other corrosive poisons. In this condition the mucous membrane is ulcerated, and the extent of these ulcerations will depend upon the severity of the caustic action; they may even cause perforation. In milder cases, where the caustic poison is not so concentrated or where the poison is taken into a full stomach which is quickly emptied by vomiting, the ulcerations may not be so extensive and the patient may recover. Recovery may be followed by a cicatricial contraction of the stomach, the pharynx or the esophagus, producing deformities with more or less stenosis of the esophagus or pylorus. A severe esophagitis and more or less pharyngitis are commonly present.

**Symptomatology.**—In ordinary cases, not corrosive, the beginning of the attack cannot be distinguished from an acute gastric indigestion, except perhaps in the severity of the symptoms. Nausea, vomiting, pain, fever and prostration mark the onset of the disease. The vomiting, which is an early symptom, is very severe; the stomach is emptied of its contents, but the nausea and vomiting continue, resulting in the expelling of small quantities of sour mucus or bilious matter which may be tinged with blood; the vomitus unlike that found in Recurrent Vomiting never contains free hydrochloric acid. The taking of food and water greatly aggravates these symptoms. Pain in the stomach is commonly present in older children, and after a few hours is followed by epigastric tenderness. The fever is especially high in infants and may within the first few hours reach 104° or 105° F., but after the stomach has emptied itself it gradually subsides. Convulsions may occur in young infants and is a dangerous complication in weaklings. Prostration is great, especially early in the disease, but gradually subsides under proper treatment. The tongue is coated, the breath is foul, and on the second or third day there is usually a complicating diarrhea. In favorable cases where treatment is begun early, convalescence should be established between the third and the sixth day. But in cases

that are badly managed and improperly fed before convalescence is established, relapses occur, and the disease may in this way be prolonged or converted into a gastroenteritis. The above clinical picture applies to infants. In older children the fever, vomiting and prostration are much less severe and convulsions rarely occur, but the gastric colic is commonly more marked.

In the CORROSIVE FORM of this disease, that produced by caustic poisons, all of the above symptoms are greatly exaggerated. The pain is intense, the vomited matter commonly contains blood, and prostration is extreme; these symptoms may continue for a number of days, resulting in death. In milder cases there is a prolonged convalescence covering weeks and sometimes months, resulting in the deformities and contractures above mentioned. In this form of the disease there is also great pain in swallowing due to the complicating pharyngitis and esophagitis.

**Treatment.**—The early treatment of the NONCORROSIVE FORM of acute gastritis is the same as that of acute gastric indigestion. The stomach may be emptied by lavage if the vomiting has not accomplished this purpose. An enema should be given and absolute quiet for the patient and rest for the stomach insisted upon. Older infants and children may get more or less comfort and satisfaction in being allowed to suck small pieces of ice held in a cloth, but no food or medication is indicated for some hours after the onset of the attack. After vomiting, however, has subsided, a dose of milk of magnesia may be given, and, if necessary, water or barley water may be given in small quantities. On the second day of the disease the diet should consist of barley water, meat juice and small quantities of Nestlé's food to which a diastase has been added. On the third day, in addition to the above-named foods, whey may be given. On the fourth day, if the child's convalescence has been satisfactory, a small quantity of skimmed milk may be added to the whey or Nestlé's food and from this time the return to cow's milk should be very gradual. If the infant happens to be breast-fed it may return to the breast milk on the third day. In very young and delicate bottle-fed infants it may be advisable to hasten convalescence by obtaining a wet nurse on the third or fourth day of the disease. Throughout the attack the bowels must be kept open; this may be accomplished by calomel, milk of magnesia and enemata. It is advisable in all conditions where the stomach is inflamed to especially avoid fats or oils either in the form of food or medicines. For this reason castor oil is not advisable as a cathartic, and cream is not to be recommended as a food until convalescence is well established.

Other medication than that above mentioned is rarely necessary in cases of simple gastritis in infants, but if the vomiting and gastric irritation persist half-teaspoonful doses of simple chalk mixture may be given at intervals of one or two hours, or compound chalk powder in 2 or 3-grain doses may be used. In older children it may be necessary to make hot applications to the stomach for the relief of pain and, after the stomach has been thoroughly emptied, to give small doses of bismuth and sometimes paregoric, put up in chalk mixture, for the relief of the pain and gastric

irritation, but the bismuth and paregoric should be dispensed with as soon as the special indications for their use have disappeared.

In CORROSIVE GASTRITIS it is absolutely necessary to empty the stomach as soon as possible with some kind of an emetic, such as the syrup of ipecac. After this has been accomplished, hot applications should be made to the stomach and morphin should be given hypodermically for the relief of pain. A solution of muriate of cocaine in 1/30-gr. doses may be given every hour for three or four hours. Following this the pain and irritation of the stomach may be relieved by chalk mixture containing small doses of paregoric or morphin, and the child must be kept alive by hypodermoclysis of physiological salt solution and by the administration through the bowel of physiological salt solution, whiskey and nutrient enemata. The stomach is to have as long a period of rest as possible. In some instances it may be necessary to abstain from food of all kinds for four or five days, allowing the child on the second or third day small quantities of water. When the gastric ulceration permits the administration of food the dietetic management of the case is the same as that just given in acute gastritis, except that one must progress more slowly with the feeding and that milk may have to be abstained from for a period of four or five weeks.

## DILATATION OF THE STOMACH

**Etiology.**—In infancy the walls of the stomach are thin and the muscular resistance slight. These anatomical peculiarities predispose to dilatation. Rickets, chronic gastritis, syphilis, anemia and general malnutrition still further weaken the walls of the stomach, which makes it possible for slight causes to produce distention and permanent dilatation. The most common exciting cause is the giving of food in large quantities at short intervals. If an infant four months of age is fed six or eight ounces of food, or all it will take, when on account of its malnutrition it should be fed only three or four ounces at intervals of three or four hours, it is evident that gastric distention and permanent dilatation may result. Chronic gastric indigestion and chronic gastric catarrh are also common causes of dilatation.

**Symptomatology.**—In the beginning, vomiting, gastric pain, tenderness and the other symptoms of chronic gastric indigestion are present, and general malnutrition, great emaciation, marked anemia and profound asthenia gradually develop.

**Diagnosis.**—The diagnosis, however, is made by the physical examination. The abdomen is distended and tympanitic, especially in the epigastric region. The quantity of food which the infant takes may indicate dilatation. When the stomach is full its lower outline may be mapped out by percussion if the infant is held in an upright position; if this dull outline reaches nearly to the umbilicus the stomach is dilated, if it reaches below the umbilicus, it is, according to Holt, much dilated. After the stomach has been washed out it may be inflated by pumping air into it with a stomach tube; in this way the distended stomach may be outlined against

the thin abdominal wall and the limits of its tympanitic note be marked out by percussion. By some one of the above methods one can, as a rule, determine whether or not the stomach is dilated, but the outlining of the infantile stomach is after all more difficult in practice than it is in theory.

**Prognosis.**—The above description applies only to simple dilatation produced by the etiological factors given above and does not apply to the forms of dilatation which occur from organic stricture of the pylorus. The prognosis, therefore, depends largely upon the severity of the underlying predisposing causes; if these are present in a marked degree it is very much more unfavorable. If, however, the constitutional disorders are not such as to produce a profound malnutrition then the prognosis under proper treatment is comparatively good.

**Treatment.**—The treatment is largely dietetic and is practically the same as that given for chronic gastritis. The food should be carefully selected to suit the individual infant; breast milk, whey and, where the child is altogether bottle-fed, small quantities of fat-free milk combined with a malted cereal mixture may be recommended. Great care as to diet, over a long period of time, is necessary to obtain success in these cases, and it is especially advisable that the food should be given at rather longer intervals and in rather smaller quantities than the weight and age of the baby would justify under normal conditions. Most important also is the hygienic management; the infant should have as much fresh air and sunlight as possible, and, if the climatic conditions are unfavorable, it should be removed to a climate where an out-of-door life is possible.

In the medical treatment hydrochloric acid and nux vomica, combined with essence of pepsin, in doses suited to the age of the infant or child, are of value. It may also be necessary to wash out the stomach at intervals and to give as a laxative occasional doses of calomel or milk of magnesia. If rickets, syphilis or tuberculosis be present they should receive proper treatment.

## ULCER OF THE STOMACH

This is very rare in childhood and even more so in infancy. The diagnosis is most frequently made on the post-mortem table, the child having died from some intercurrent disease. From these autopsy findings it is evident that the disease is not commonly characterized by a symptomatology sufficiently definite to make the diagnosis plain. When these ulcers occur, as they sometimes do in connection with acute gastritis, we may have marked gastric irritation with nausea and vomiting; when there is a bloody vomitus, associated with tarry stools, the diagnosis is commonly simple enough. Rotch and others have recorded cases of simple perforating ulcers, but these cases are extremely rare. Tuberculous gastric ulcers are also seldom seen.

**Treatment.**—In severe cases rectal feeding may be necessary and opium may be required for the relief of pain, but for the most part the treatment consists in a preliminary rest for the stomach, followed by a carefully regulated diet, such as is necessary in acute gastritis. The use of alkalies,

such as lime water and sodium bicarbonate, and stomach sedatives, such as bismuth, are of value in relieving the symptoms. If perforation occurs laparotomy is to be resorted to at once.

## ACUTE GASTRODUODENITIS

(*Catarrhal Jaundice*)

Catarrhal jaundice occurs usually between the second and the fifth year, being almost unknown in infancy and uncommon after the fifth year. It is probable that all cases are due to some infection, which produces more or less duodenal catarrh with an accompanying catarrh of the common bile duct, resulting in its obstruction. The bacteriology of this condition is not known, and at the present time it seems improbable that all cases are due to the same bacterial infection. It is much more probable that the disease may be produced by a number of infections, prominent among which is influenza. The writer on a number of occasions has seen two cases in the same family of children, where there was a house epidemic of influenza.

**Symptomatology.**—The early symptoms are those of mild gastric indigestion, nausea, vomiting, gastric discomfort, fever from 100° to 103° F., more or less headache, irritability, nervousness, mental depression and general discomfort. These symptoms continue for three or four days and then jaundice appears and the diagnosis is made. The skin and conjunctiva assume a yellowish hue which gradually deepens to a saffron tint. The child is usually constipated, the stools are white or clay colored and have a bad odor. The urine contains bile and is of a yellowish-brown color. There are pain and tenderness over the duodenum, the tongue is heavily coated, there is much thirst and little appetite, and occasionally there is an uncomfortable itching of the skin, but this latter symptom is not so common as in the forms of chronic jaundice seen in the adult. The liver is almost always enlarged and remains so for some days after the jaundice has disappeared. The spleen may be palpated in most cases.

**Course.**—The disease lasts from two to three weeks, and the symptoms are so uniform and the course so definite that it gives one the impression of a self-limited acute infection. In the ordinary course of the disease the gastric symptoms subside within the first week, the temperature becomes normal, the appetite returns, and the general discomfort disappears, but the jaundice, with the clay-colored stools and bile-colored urine, continues to the end of the third week, when convalescence is usually established, but it may be delayed by errors in diet.

**Diagnosis.**—In early infancy catarrhal jaundice does not occur. The differential diagnosis from other conditions producing a sallow skin is made by the yellowness of the conjunctiva and by the bile in the urine.

**Treatment.**—Preliminary to the onset of the jaundice, before the diagnosis is made, the child is treated for ordinary gastric indigestion. With the onset of the jaundice calomel is to be given, followed by Rochelle salts, and throughout the course of the disease the bowels are to be irrigated every

day or every second day, and phosphate of soda is to be given in sufficient quantities to insure slight laxative action. The diet during the acute stages of the disease should be of skimmed milk, buttermilk, bread, cereals, broth and a little orange juice. Fats are to be especially avoided and meats are to be given sparingly, since the bile, as the author [1] many years ago demonstrated, is necessary for the ready digestion and assimilation of both fat and protein. The cereals are very well borne, especially if a diastase be given with the meals.

## CONGENITAL HYPERTROPHY OF THE PYLORUS

This condition has attracted considerable attention in recent years, and there can be no doubt but that the clinical syndrome embraced under this heading is not so infrequent as formerly supposed, but there is still much difference of opinion as to the actual lesions which produce it.

**Etiology and Pathology.**—It is generally conceded that there is a congenital condition in which the pylorus is thickened and its lumen so greatly

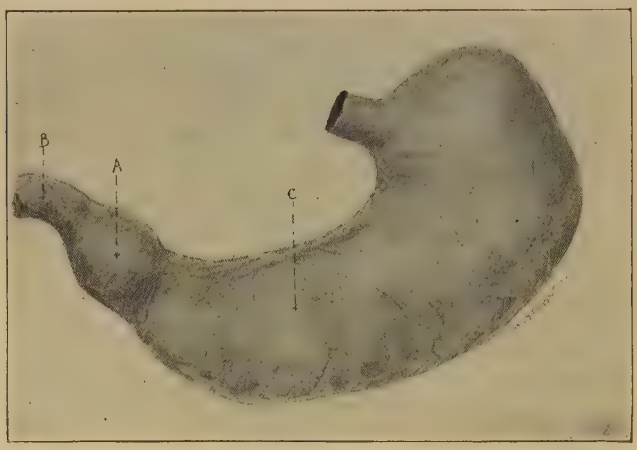



Fig. 25.—Congenital Stenosis of the Pylorus. (Bevan.)

reduced, that it acts as a serious obstruction to the transfer of the food contents of the stomach to the small intestine. The pylorus may form a small tumor which can be felt and occasionally seen through the abdominal wall. The lumen at the point of greatest hypertrophy of the sphincter may be so small as to admit only a fine probe. It is also conceded that this muscular hypertrophy, which involves the pyloric end of the stomach as well as the pylorus itself, is associated with spasm, which greatly aggravates the constriction. Thompson suggested that the pyloric spasm might be the initial lesion occurring soon after birth, being produced by the swallowing of liquor amnii, and that this fluid and other irritating substances produced by the fermentation of food and mucus might continue the exaggerated

[1] *Journal of Physiology*, 1891.

action of the pyloric sphincter and muscular coats of the stomach, and thus produce a secondary hypertrophy of these muscles, which would increase the constriction and finally result in more or less marked hypertrophy and permanent constriction. Whatever may be the relative importance of the congenital hypertrophy and the pyloric spasm in the etiology of this condition, it is conceded that both these factors exist, and I am inclined to believe with Koplik that there are two distinct groups of cases: one in which there is a spasm of the pylorus and pyloric end of the stomach, with little or no hypertrophy, and the other in which the essential lesion is hypertrophy of the pylorus, and that the symptom group in these cases is aggravated by an associated pyloric spasm.

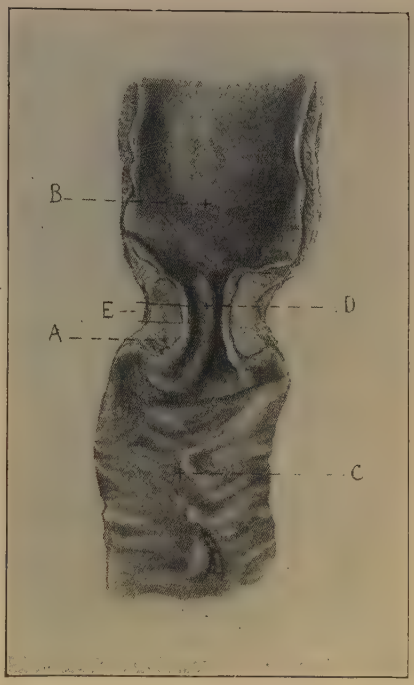



FIG. 26.—CONGENITAL STENOSIS OF THE PYLORUS.

Longitudinal section, through tumor mass. (Bevan.)

**Symptomatology.**—The first and all-important symptom in these cases is vomiting. In the great majority of cases it begins in the third or fourth week, but in rare instances it may begin a few days after birth or may be delayed to the seventh or the eighth week. In the beginning the vomiting may occur only once or twice in the twenty-four hours, but gradually increases in frequency and force, until, within the course of a week or ten days, nearly all of the food is vomited directly after taking. In the rapid development of this symptom there can be little doubt but that pyloric spasm plays an important rôle, and even during the height of the disease there is at times a strange intermittency in the force and frequency of the vomiting. Instead of occurring with great force directly after the taking of food this character of vomiting may be superseded for a few hours by comparative tolerance on the part of the stomach for food, and the vomiting may occur only after the food has remained in the stomach for hours, and again, directly after vomiting, food may be taken and retained until after the next feeding, when the whole contents of the stomach are ejected with considerable force. The infant ceases to gain in weight and then commences to lose and, as the chronic vomiting continues, becomes emaciated and malnourished to the last degree. Constipation is always present; it may exist with a discharge from the bowel several times during the day of small liquid stools, consisting largely of mucus and bile. A close study of these

discharges will show that comparatively little or no food has passed from the stomach into the intestine. After taking food the child suffers more or less gastric discomfort, and if the disease continues for a long time, there is more or less marked dilatation of the stomach. It is an afebrile condition.

All of the above symptoms may be produced by pyloric spasm even though there be little or no hypertrophy of the pylorus, but when marked hypertrophy of the pylorus exists the above symptom group is exaggerated, and there are in addition two physical signs of great diagnostic importance. One of these, the most characteristic of all, is a small, movable nodule which may be felt on deep pressure in the region of the pylorus, between the margin of the liver and the umbilicus. This tumor, which should always be searched for, is the pathognomonic sign of hypertrophy of the pylorus, and in cases where it cannot be felt there must remain some doubt as to the presence of this condition. The other sign which is commonly noted in these cases is the peristaltic movements of the stomach, which can be seen through the distended abdominal wall. Valuable information as to the patulency of the pylorus may be obtained by giving the infant a large dose of subnitrate of bismuth, and one hour later determining by an X-ray picture whether or not the bismuth has passed the pylorus.

**Diagnosis.**—According to Koplik, the diagnosis of pyloric spasm without hypertrophy of the pylorus is made largely by the absence of the pyloric tumor, and the presence of the stomach peristalsis and a projectile vomiting which empties the stomach, and the presence in the stools of a certain amount of fecal matter, which indicates that considerable food may have passed from the stomach into the intestine. The diagnosis of hypertrophy is made by the presence of all the symptoms of pyloric spasm, plus the pyloric tumor, the stomach peristalsis, the almost complete absence of fecal matter in the stools and retentive vomiting in contradistinction to full vomiting. Morse says, the most important points in favor of spasm in doubtful cases are the absence of a palpable tumor, or, if a tumor is present, its cord-like feel, the presence of intermittent contraction and relaxation of the tumor, and rapid improvement under medical treatment and regulation of the diet. He believes that a cord-like tumor may sometimes be felt when no hypertrophic stenosis exists, and that in some cases of hypertrophic stenosis no tumor can be felt. He also says that if the baby is breast-fed the chances are greatly in favor of hypertrophic stenosis, and if it be artificially fed, the chances are even.

The only common disease with which hypertrophy of the pylorus may be confused is *chronic gastric catarrh*. This condition may be differentiated by the fact that in chronic gastric catarrh there is apparent cause for the onset of the vomiting and also for its continuation, and the vomiting in this condition subsides when the stomach is rested and a proper diet is given, so that there is little reason for mistakes in diagnosis after the conditions have been studied for a few days.

There are other very rare conditions, such as stricture of the duodenum, either congenital or resulting from cicatricial contractions following ulcers,

and scars of the pylorus, which are so infrequent that they scarcely deserve consideration. In the event, however, of the existence of these conditions their treatment would be the same as that of congenital hypertrophy of the pylorus.

**Prognosis**—The prognosis of pyloric spasm uncomplicated by hypertrophic stenosis is favorable. But the prognosis of pyloric stenosis, which is always complicated by pyloric spasm, is grave, since many of these cases are not relieved by dietetic treatment. The surgical treatment, however, is successful in about 50 per cent. of these cases, and the after results are, as a rule, good, although Koplik notes that quite a percentage of the cases which survive the operation ultimately develop grave forms of malnutrition. The fact remains, however, that in a large percentage of these otherwise hopeless cases, surgery saves the life of the child and gives it a fair chance to be restored to a satisfactory condition of health. Of the five operative cases that have come within my personal knowledge, four recovered and are now apparently well.

FIG. 27.—PYLORIC STENOSIS. (Radiograph.)
Taken three-quarters of an hour after the administration of bismuth paste. Note fine stream of paste escaping from the pylorus. Patient age thirteen weeks. Complete recovery without operation. (Max Dreyfoos.)

**Treatment.**—In view of the fact that a large percentage of these cases may be cured without resorting to the knife, it is advisable that every case should have careful dietetic and medical treatment until it has been fully demonstrated that such treatment is of no avail. Since all of these cases occur so early in life, the great majority of them are developed on

breast feeding, but, whatever may be the food of the infant, with the onset of the symptoms the stomach should be washed out with a sodium chlorid solution (one level teaspoonful to a pint of water), and throughout the treatment, especially where retentive vomiting is present, stomach washing should be resorted to, from time to time, as necessary. After the preliminary washing of the stomach no food should be given for a period of at least twenty-four hours, but during the latter portion of this time small quantities of salt solution may be given by the mouth. If this is retained two or three small breast feedings may be given within the next twenty-four hours. If the breast milk disagrees and the vomiting recurs, the stomach should have a rest and the breast milk of another wet nurse may then be tried. Skimmed breast milk will sometimes be retained when ordinary breast milk will not. It is important that the breast milk be given in small quantities and at intervals of at least four hours during the first three or four days of the treatment; in some instances the gastric irritation and pyloric spasm may be relieved by giving directly before each nursing a few teaspoonfuls of equal parts of water and lime water. During this period of insufficient nourishment small quantities of salt solution may be given by the mouth, and 5 or 6 ounces of the same solution may be thrown high up into the bowel at such intervals as may be necessary to overcome thirst and to supply the body with fluids. If breast feeding fails entirely a radical change in the food may be resorted to. Whey and peptonized fat-free milk are foods which may serve the temporary purpose of allaying gastric irritation and pyloric spasm, and thus pave the way for a third trial of breast milk. If success follows any form of dietetic treatment one should be most careful not to change the diet of the infant until the condition of the child's stomach warrants, and the nutrition of the infant demands that a change should be made. In the event, however, that all dietetic measures fail, the patient should be prepared for operative treatment by hypodermoclysis of physiological salt solution and turned over to the surgeon for operation. He may then determine the nature of the operation to be performed in the individual case. Gastroenterostomy, pyloroplasty and Loreta's operation have all been performed with success in these cases; the first of these is the favorite at the present time.

## CHRONIC GASTRITIS

(*Chronic Gastric Catarrh*)

This is a very common disorder, especially in infants and young children, and is usually associated with diarrhea and chronic intestinal indigestion; this subject will be more fully treated under the latter heading.

**Etiology.**—Chronic gastric catarrh is frequent during the first year of life and is usually caused by taking too much food at too short intervals; this applies especially to bottle-fed babies. If for example an infant three months of age, that should be taking 4 ounces of food every three hours, is given 6 or 7 ounces or "all it will take," especially at irregular intervals,

trouble will surely follow. The overworked stomach becomes distended, its motor power diminished, its mucous membrane diseased, the gastric secretions impaired, but appetite and thirst may still continue, so that the infant's stomach is never empty, and fermentation and gastric irritation result; such is the common etiology of this disease in young infants. Bad hygiene, impure air, lack of sunlight, filthy surroundings and constitutional diseases, such as tuberculosis, syphilis and rickets, which produce malnutrition and anemia, are important predisposing causes. Recurring attacks of acute gastritis may be a part of the early history of this disease. In older children also, the cause is too much food and improper food at irregular intervals.

**Pathology.**—The stomach is dilated and the mucous membrane is covered with a tough tenacious mucus. The changes are somewhat similar to those found in acute gastric catarrh, except that the swelling and congestion of the mucous membranes are not so marked, and petechial hemorrhages and marked injection of blood vessels do not exist. The mucous membrane is infiltrated with round cells, is thickened, and there are erosion and degeneration of the epithelial cells, especially in and around the gastric tubules.

**Symptomatology.**—Vomiting is the characteristic symptom. It may occur every day, or every other day, or at longer or shorter intervals, depending upon the severity of the case and the character of the food administered. In older children vomiting occurs more frequently in the early morning than at any other time, but in infants it may occur at any time when the stomach is overfull. The vomited matter consists of undigested food and of glairy, tenacious mucus which is acid from the presence of the fermentation acids, acetic, butyric and lactic; hydrochloric acid is almost never present. In infants gastric indigestion and diarrhea are common; children over two years of age are, as a rule, constipated. For a long time the appetite may remain good and is unfortunately much larger than the digestive capacity. The child is nervous, irritable, sleeps badly, frets, whines and demands more or less constant attention. It may cry for food and take with avidity more than the normal quantity, and yet suffer from gastric pain and discomfort soon after. It fails to gain in weight and as the disease progresses there are loss of weight, dry skin and anemia. The face becomes thin and old-looking, the belly large and tympanitic and the legs thin; the appetite is gradually lost, and emaciation and malnutrition increase until death occurs from exhaustion. This extreme picture, however, is fortunately not very common except in young infants. In older infants and children all of the symptoms are much less severe, the vomiting is not so frequent and the disease yields much more readily to treatment, so that, as a rule, the severe symptoms leading to extreme emaciation and profound malnutrition are uncommon unless the intestinal canal be involved.

**Diagnosis.**—The diagnosis is not difficult, the chronic vomiting, gastric discomfort, epigastric tenderness associated with malnutrition and anemia, occurring as an afebrile condition in an infant, cannot be mistaken for any

other disease except congenital pyloric spasm. (See Congenital Hypertrophy of the Pylorus.)

**Prognosis.**—The prognosis largely depends upon the age of the child and the stage of the disease when proper treatment is instituted. If the diagnosis is made early the prognosis is good even in young infants, but otherwise at this age it is very doubtful. In children over three years of age it is good, but it may require months or years in a well-established case to completely overcome all evidences of the disease.

**Treatment.**—One of the first indications is to cleanse the stomach and keep it as clean as possible. In infants under one year of age this may very readily be done by washing out the stomach once a day with a weak bicarbonate of soda or sodium chlorid solution, one-half drachm to the pint; this warm alkaline solution should be used about three hours after a feeding. In older children the stomach tube cannot so readily be used and in these cases the cleansing may be done by milk of magnesia, Rochelle salts or sodium phosphate, taken before meals once or twice a day in sufficient quantities to relieve constipation. If the children are old enough it is also advisable to have them sip hot water in which has been dissolved some bicarbonate of soda; this should be done one hour before meals. The careful selection of a proper diet is very difficult in these cases, and yet upon it depends their successful treatment. On general principles one may say it is advisable at all ages to avoid fats and to give small quantities of food at long intervals, the object being to have the stomach empty itself before it is required to undertake the digestion of another meal. The diet must, of course, suit the age and digestive capacity of the individual infant. If the food happens to be breast milk, then something must be radically wrong with that particular breast milk, or it must have been fed as to interval and quantity most unwisely. In such instances it is advisable, if the symptoms do not yield readily, to change to another wet nurse, and if this procedure fails the infant should be put upon modified cow's milk. Infants that have been taking cow's milk or other foods when the gastric catarrh developed are to have their milk formulas carefully regulated with reference to their digestive capacity. The foods that usually agree with these infants, fat-free milk, peptonized milk and buttermilk, may be tried in the order named. The fat-free milk and the peptonized milk should be diluted with dextrinized gruels, and, as the infant improves, dextrinized gruels may be diminished and the quantity of skimmed milk or peptonized milk in the mixture gradually increased. The buttermilk mixture may be prepared by adding to a pint of buttermilk a tablespoonful of wheat flour and two tablespoonfuls of cane sugar, and boiling thirty minutes. This mixture may be given every four hours to infants between the ages of six and ten months, and the quantity should be one ounce less than they are months old. This is an excellent food and agrees with many cases. Meat juice, in suitable quantities, diluted with a little water, may be added to the infant's diet after it has been demonstrated that any one of the above-named milk preparations is agreeing with it. One of the proprietary milk foods, such as

Nestlé's, is often of value in the treatment of these cases. If the Nestlé's food is used, 5 or 10 drops of a liquid diastase should be added to each feeding and later small quantities of fat-free or peptonized milk are to be added, and as these foods are added the proportion of the Nestlé's food is diminished, so that in time one of the milk preparations gradually replaces the proprietary food. In older children, as they commence their convalescence, meat, eggs, cereals, bread and later orange juice may be added.

The hygienic treatment of these cases is very important; it may be necessary to send them to a cool climate during the hot summer months and to a warm climate during the cold and disagreeable months of winter. The object of these changes is to put the infant under such climatic conditions as will give it plenty of sunlight and fresh air without subjecting it to the depressing effects of the heat of summer or the chilling effects of damp, cold winters. If it be necessary to keep it out of doors in cool weather it must, because of its diminished vitality, be dressed warmly; above all, the feet and legs should be kept warm by hot water bottles and proper clothing. In young infants inunctions of lanolin once a day serve the purposes of furnishing a light form of exercise, increasing the peripheral circulation and causing the absorption of small quantities of easily assimilable fat. In older children mild exercise in the open air is advisable. Hydrochloric acid and pepsin given after meals are of value in many of these cases. Pancreatin and the thick malt preparations may be used to promote the digestion and the absorption of carbohydrate foods. In older children nux vomica, in small doses before meals, serves to stimulate the digestive capacity. Holt recommends the use of salicylate of soda in 1- or 2-gr. doses to control gastric fermentation.

## CHAPTER XIX

### ETIOLOGY AND PREVENTIVE TREATMENT OF THE INTESTINAL DISORDERS OF INFANCY

### ETIOLOGY

These disorders include intestinal indigestion, intestinal intoxication and intestinal catarrh. To avoid repetition the etiology and preventive treatment of these conditions may be considered under the same heading, since they very commonly occur in the sequence given above. The indigestion may be the initial disturbance which predisposes to the intoxication, and the latter very commonly results in intestinal catarrh. These intestinal disorders are not sharply defined from each other either in their etiology or treatment. On the other hand, they are to a large extent the result of the same etiological processes and commonly but different steps in the same pathological process, and their treatment is very much along similar lines. Kerley has justly laid great stress upon the importance of intestinal indi-

gestion in predisposing infants to more serious gastrointestinal diseases, and German writers, especially Czerny, Keller and Finkelstein, have demonstrated that the most serious gastrointestinal disorders follow in the wake of indigestion produced by overfeeding. The relationship which exists between intestinal indigestion, intoxication and catarrh is forcibly brought to our minds by the increased death rate of infants during the hot summer months. Infants who have suffered from frequent attacks of gastrointestinal indigestion throughout the year and who have, therefore, feeble digestive capacities and feeble powers of resistance, are very prone to diarrheal diseases. Infants of this class are often wrongly judged to be incapable of digesting cow's milk and are, therefore, injudiciously placed upon more easily digested foods, such as condensed milk, proprietary foods and other ill-balanced food formulas, and as a result of such feeding they become rachitic and otherwise malnourished. With these malnutritions superimposed upon a feeble digestive capacity and feeble powers of intestinal resistance, they are ill prepared to stand the dangers from more serious gastrointestinal diseases which beset them during the hot summer months. The great death rate among weaklings of this class perhaps accounts for the rapid increase of infantile mortality from diarrheal diseases in our large cities during the early summer months.

Among the predisposing causes of the intestinal disorders of infancy are rickets, syphilis, tuberculosis, anemia, neurotic inheritance, prolonged heat of summer and bad hygienic conditions, including impure air and lack of sunlight. Any or all of these factors may, by lowering the resistance of the infant, reduce its physiological digestive capacity, diminish its tolerance for any one of the food ingredients of milk and predispose it to attacks of indigestion, intoxication and intestinal catarrh. A feeble, malnourished infant that has been getting on fairly well on a carefully modified milk mixture may, when the heat of summer lowers its digestive capacity, suffer an acute intestinal disturbance caused by the self-same food upon which it has been previously thriving. In normal infants a much more potent exciting cause, such as *overfeeding* or contaminated milk, is commonly necessary to produce intestinal disorders.

Intestinal disturbances are much less common and much less severe in breast-fed than in artificially fed infants. During the first week of life indigestion is common, resulting from the physiological incompetency of the gastrointestinal canal and abnormalities in the mother's milk; it, as a rule, quickly disappears as the intestinal canal adjusts itself to its physiological duties and the milk supply of the mother becomes more normal and stable in its composition. Later on in the life of the infant, indigestion and diarrhea may result from variations in the fat and protein constituents of the milk, produced by menstruation, nervous shock, dissipation, imprudence in diet, ill health and the failure to observe proper hygienic rules on the part of the mother. These defects are usually transitory and can be corrected by regulating the life and diet of the mother; if not, there is some radical defect in the milk and another wet nurse should be secured. There is,

however, no more common error in infant feeding nor one that is responsible for greater loss of life than that of taking infants from the breast for slight and remediable causes.

*Overfeeding* is the most important cause of indigestion, and the indigestion thus produced may lead to more serious intestinal disorders; this fact has been most graphically pointed out by Czerny and Keller. Very pronounced and very persistent gastrointestinal indigestion may result even from normal breast milk given in *too large quantities* and at *too short intervals;* but it must be said that the infant shows a remarkable tolerance for human milk; it is more difficult to make it ill by overfeeding with breast milk than with any other food. The breast milk, however, taken in twenty-four hours should not, in caloric value, much exceed the demands of the infant, and it should not be given at such short intervals that the stomach will not have time to almost or quite empty itself before another supply enters it. In America the dangers from overfeeding with breast milk are not very great, since American mothers, as a rule, furnish insufficient rather than a superabundant supply of milk. This form of indigestion, if recognized, is easily corrected in the breast-fed infant by allowing smaller quantities of breast milk at longer intervals until convalescence is established and then allowing it to nurse at regular four-hour intervals.

In artificially fed infants *overfeeding,* as a cause of intestinal disorders, is much more important and much more serious in its consequences. It may produce violent and prolonged gastrointestinal disturbances, which may be complicated by intestinal infection and end in enterocolitis. Czerny, Keller, Finkelstein and others insist with reason that, apart from a complicating infection and intestinal catarrh, overfeeding may produce an injury to the metabolic processes of the infant, manifesting itself at first in a severe indigestion and later in fever and profound nervous and other constitutional symptoms so severe that it may require weeks of careful underfeeding to restore the infant to a normal condition. In most of these cases it will be found that the infant has suffered a special "food injury" which very markedly diminishes its tolerance for either the fat, the sugar, the salts or the proteins of milk, and, in the subsequent feedings, it will be necessary for a time to greatly reduce the particular ingredient of the milk for which the infant's tolerance has been reduced; this is commonly, early in the disease, the fat (cream), and later, the sugar or whey salts. Overfeeding is a much more potent cause of indigestion if it be associated with too frequent feedings; the digestive organs of the artificially fed infant must have regular periods of rest, and this means feeding at long and at regular intervals. Too rapid increase in the strength of the food formula and improper and unwholesome food are important causes of indigestion. In the artificially fed infant, whatever may be the cause, the results are less serious if it can be fed during its convalescence on breast milk. These dietetic errors may produce acute intestinal disorders even in normal infants, but they act much more rapidly and the symptom complex which they produce is much more severe in feeble, malnourished children.

The *swallowing of mucus,* which occurs in catarrhal conditions of the respiratory tract in infants, is a very common cause of gastric and intestinal disturbances. Unwise cathartic medication or drugs, especially those belonging to the so-called expectorant class, such as ipecac, squills and ammonia, so commonly given to very young infants suffering from catarrhal conditions of the respiratory passages, may, if not given with much discretion, produce acute intestinal disorders. *Dentition* and exposure to wet and cold may, especially in feeble infants, act as exciting factors of indigestion and diarrhea.

In older children much more potent factors than those above named are commonly required to produce attacks of intestinal disturbance. They are not so easily upset by the quantity of food taken or by eating at irregular times provided the food is wholesome, nor are they so easily affected by the ordinary bacterial contamination of milk. Indigestion with them commonly results from rather gross errors in diet, such as the eating of green fruit and large quantities of sweets and pastries.

Acute intestinal toxemia may come and go without producing acute enteritis, but on the other hand practically every enteritis is preceded or accompanied by a *bacterial infection* producing an intestinal toxemia and subsequent catarrh. This, however, does not imply that the initial or most important etiological factors of every case of enteritis are *bacteria,* but it does imply that the bacterial factors are all-important in producing the pathological changes which underlie and prolong these diseases. Acute intestinal toxemias are caused by a variety of microorganisms which produce, by their action on food stuffs, soluble and irritant poisons. Some of these poisons irritate the intestinal mucosa; others are absorbed and exert a poisonous action on the nerve centers, especially the anterior horns of the spinal cord. In some instances the poisonous bacterial products are formed in such quantities in milk and other food before they are taken into the gastrointestinal canal, that a violent and dangerous intoxication follows directly upon taking such contaminated food. These cases are commonly grouped under the term "milk or food poisoning," and they have been especially elucidated by the researches of Vaughn, who has succeeded in isolating from contaminated foods soluble poisons, among them tyrotoxicon, by the introduction of which into the gastrointestinal canal of animals he was able to produce a symptom group similar to that produced by food poisoning. In other instances—and these are the common ones—the milk at the time of taking is so slightly contaminated with pathogenic bacteria that it simply acts by starting a pathogenic fermentation in the food contents of the intestinal canal of the infant. As this fermentation proceeds, irritant and soluble toxins are formed, which sooner or later produce more or less severe symptoms of intestinal irritation and constitutional poisoning. It is also probable that certain bacteria which are commonly present in the intestinal canal of normal infants may, as a result of overtaxing the digestive organs with too much food or improper food, become pathogenic, setting up abnormal fermentations which produce irritant and constitutional poisons.

Since milk is the great carrier of pathogenic bacteria and their soluble toxins into the intestinal canal of the infant, it follows that contaminated milk is by far the most important cause of gastroenteric intoxication and that all of the conditions therefore which predispose to the contamination of milk are important etiological factors of this condition. In laying stress upon the important rôle which milk plays as a carrier of pathogenic bacteria, it should not be overlooked that such bacteria may find an entrance into the intestinal canal of the infant in other ways; in the water it drinks, on the foreign bodies it puts into its mouth and more especially in the mucus which it swallows. I have been much impressed in recent years with the fact that too little stress has been laid upon this latter form of contamination. In catarrhal processes of the nose, pharynx and upper air passages which occur in the acute infections, large quantities of mucus are secreted and swallowed by the infant. This mucus contains large numbers of streptococci, staphylococci, influenza bacilli and other microorganisms, which may infect the intestinal mucosa or produce a pathological fermentation of food stuffs in the intestinal canal, thereby producing an enteric intoxication. During the winter season this is perhaps the most important cause of this condition; the cases of so-called intestinal grippe and septic enteritis follow in the wake of these intoxications.

Notwithstanding the fact that we speak with such confidence as to the rôle which bacteria play in the production of acute gastroenteric infections, yet it must be admitted that, in spite of the enormous amount of work that has been done by bacteriologists in the study of the normal and pathological intestinal flora, we have not as yet been able to associate definite gastroenteric infections with specific pathological microorganisms, nor is it possible for us to say that the normal intestinal flora may not under pathological conditions play a rôle in producing these conditions. The intestinal canal of the newly born infant is free from microorganisms. Within a few hours after birth, however, the normal intestinal flora begin to make their appearance, and the character of microorganisms present will depend upon the food of the infant. If the infant is breast fed the prevailing types are the bacillus bifidus and bacillus acidophilus. The acidouric group, which flourishes in carbohydrate media, predominate until the food of the infant is changed to cow's milk or until some other albuminous food is added to its diet. These acid-forming bacilli protect the nursing infant against attack from putrefactive organisms. With the change to cow's milk and albuminous food putrefactive bacteria of the colon group having proteolytic action make their appearance and in part replace the acid-forming group; this change makes the child more susceptible to intestinal infection with pathogenic microorganisms. The important fact to be borne in mind is that the bacteria which inhabit the normal intestine serve a useful purpose in the digestion of food stuffs, but the most important rôle that they play is in preventing the infection of the intestine with pathogenic bacteria. The acid fermentations prevailing in the normal infant's intestines have a tendency to prevent and destroy putrefactive processes. It is also important to

bear in mind that the putrefactive processes carried on by pathogenic bacteria in the intestinal canal of the infant may be modified and sometimes controlled by a diet poor in albumin and rich in carbohydrates, the carbohydrate foods favoring the development of some of the normal acid-forming intestinal bacteria which have a tendency to destroy the putrefactive bacteria flourishing in the proteins. This explains in part the value of carbohydrate foods in beginning the treatment of intestinal indigestion; the temporary success which follows the use of condensed milk, Nestlé's food and other foods of this class may be explained in this way.

Among the pathogenic bacteria which may produce intestinal infection the streptococcus enteritidis deserves special mention, and Booker is entitled to great credit, since he was the first to call attention to the rôle which this microorganism plays in this condition; he found it not only in the stools, but also in the intestinal canal and in the walls of the intestine, and in the various organs of infants who had died from acute enteric infection. Escherich confirmed Booker's observations and found this streptococcus to be the cause of epidemics of this disease, but notwithstanding these observations there is no clinical picture which can be definitely associated with streptococci. In these conditions it is believed that various species of streptococci are active. In other epidemics the staphylococcus pyogenes aureus and albus predominate. Booker also, in these early valuable researches, called attention to the proteus vulgaris as a cause of enteric infection. It was associated especially with foul-smelling, constipated, grayish stools covered with mucus. Brudzinski later observed that this organism disappeared from the intestine when milk foods were stopped and carbohydrates were given, and he also found that the same result could be obtained by inoculating the food of the infant with fresh cultures of bacillus lactis aerogenes. This is an example of controlling a pathological fermentative process by the introduction of bacteria belonging to the normal intestinal flora. Escherich, who is one of the most valued workers in this field, describes a "blue bacillus," which he believed to be the etiological factor in a severe epidemic of this disease. He also believes, with a number of other investigators, that the bacillus coli communis may produce intestinal infection. The ameba coli is associated with pathological processes in the infant similar to those found in the adult. The influenza or Pfeiffer bacillus is now generally recognized as one of the common causes of intestinal infection, especially during the winter months. This bacillus may produce a more or less severe catarrhal condition of the intestinal mucosa which is commonly known as intestinal grippe. The bacillus Welchii, or gas bacillus, is believed to be commonly associated with putrefactive intestinal disturbances.

In recent years the Shiga bacillus has been definitely associated with the etiology and pathology of gastroenteric infections both in the infant and in the adult. This bacillus, named for its Japanese discoverer, was demonstrated to be the causative factor of epidemic dysentery in the adult. Flexner and his associates in this country, and a large number of observers

the world over, have shown that the Shiga bacillus plays a pathological rôle in the gastrointestinal diseases of infancy, but this bacillus is not associated definitely with any distinct symptom group. It has been found in cases of gastroenteric infection and in mild and severe cases of enterocolitis. The more recent investigators, however, believe that it is very definitely associated with the pathological processes in acute enterocolitis in which the stools contain blood and mucus. In some of these cases it is possible to demonstrate the specific agglutinin reaction to the Shiga bacillus in the blood of the patient. This reaction, it is assumed, definitely associates the Shiga bacillus with the pathological process in the intestine. Flexner and his associates determined that there were two varieties of the Shiga bacillus. One of these, the true Shiga bacillus, is spoken of as the alkaline type; it does not ferment in mannit media; the other, the Flexner or acid type, does ferment in mannit media, and of the two it is more closely associated with *infantile* enterocolitis. But in this disease it is now recognized that streptococci and the colon bacillus also enter into the pathological process.

The infectious nature of the diarrheal diseases of infancy should be insisted upon in order to insure proper care in handling the intestinal discharges. While it is true that there is comparatively little danger that bacteria, producing intestinal disorders, will pass directly from one infant to another, it is also true that the careless handling of the intestinal discharges may so contaminate the surroundings of the infant suffering from diarrhea that other infants living in the same room will be in great danger of gastrointestinal infection. In hospitals and tenement houses infection may be a potent factor in spreading the diarrheal diseases of infancy.

*Age* is the all-important predisposing cause of the intestinal disorders of infancy. The vast majority of these cases occur during the first or second year of life. After the second year the predisposition to these conditions so rapidly diminishes with the age of the child that they are comparatively infrequent, and when they do occur are much less serious in character. When it is realized that children three or four years of age, taking the same food and living under the same hygienic surroundings, are comparatively exempt, the importance of age as a predisposing factor becomes apparent. This susceptibility on the part of infants to gastroenteric diseases perhaps may be accounted for by their lack of resistance to fermentative processes in the intestinal canal and to the resulting catarrhal processes which follow these fermentations, and by their greater susceptibility to the action of soluble bacterial poisons. These soluble poisons, acting upon the undeveloped and immature nervous system of the infant, produce high fever, convulsions, and other severe constitutional symptoms, which the better-balanced nervous system of the child resists to such an extent that these toxic symptoms are comparatively slight. It follows, therefore, that even normal infants should be protected in every possible way from all the exciting and predisposing causes of gastroenteric diseases.

*The heat of summer* is such an important factor in producing the intestinal disorders of infancy that these conditions are not uncommonly

spoken of as "summer complaint." Infant mortality, so enormously increased during the hot summer months, is largely due to the prevalence of gastroenteric diseases during that period of the year. Summer heat promotes food contamination; among the poor of our large cities who have not the means to procure clean cow's milk, nor the facilities for keeping it clean even if it were furnished them, milk is so rapidly contaminated by bacteria that it soon becomes an unsafe food for infant feeding, and is therefore responsible for a large percentage of the cases of gastroenteric infection. Even among the well-to-do, who have the facilities for obtaining and caring for clean milk, the difficulties which prolonged hot weather adds to the care of keeping milk wholesome make milk contamination and the resulting intestinal disorders of not uncommon occurrence. The heat of summer also acts directly on the infant, diminishing its digestive capacity and its normal resistance to these diseases. It is also probable, as Forchheimer has taught for many years, that many of the cases of so-called gastroenteric intoxication are due directly to the effect of heat. That is to say, the infant primarily suffers a heat-stroke, with high fever, great prostration, and secondarily an acute intestinal disorder; the latter condition continuing after the infant has recovered from the primary effects of the heat stroke.

Humidity or the amount of rainfall, according to Seibert and others who have investigated this subject statistically, has little to do with the mortality of this disease, and it is difficult to see how this cause could act deleteriously except, perhaps, in housing infants in unhygienic quarters on rainy days, and this might easily be offset by the fact that rain cleans the air and streets and reduces the temperature.

*Bad hygienic surroundings* is an important cause of gastrointestinal disorders in infancy. This fact is brought home to us by the enormous death rate of infants among the tenement house population of our large cities during the hot summer months. Infants who must pass their nights in ill-ventilated, unclean rooms and their days in the surrounding dirty streets, have comparatively little chance to escape the dangers of food contamination. Bad hygienic surroundings not only enormously increase the danger which surrounds these infants by increasing their opportunities for infection, but also predispose them to gastroenteric diseases by reason of the fact that they have lived throughout the year in close, badly ventilated quarters, with little *sunlight and fresh air,* and have therefore feeble digestive capacities and diminished powers of resistance.

## PREVENTIVE TREATMENT

Since the acute intestinal disorders of infancy are the great causes of mortality during this period of life, every infant should be cared for with special reference to the prevention of these diseases. In accomplishing this end the physician's prime object will be to place the infant upon the most available food for strengthening its digestive capacity and improving

its nutritional condition. All breast-fed babies should, if possible, be kept upon breast milk as an exclusive food during the hot summer months, and, if the breast milk be insufficient for this purpose, mixed feeding, as fully outlined in a previous chapter, should be resorted to. By this method the infant takes sufficient modified milk, following a number or all of the breast feedings, to supply its nutritional demands. The importance of a little breast milk to assist in the digestion of the cow's milk and to maintain the normal intestinal flora is of special importance during hot weather. Artificially fed children should be even more carefully fed according to the principles outlined under infant feeding. Well infants should be fed at regular intervals on a food formula suitable to their age, weight and digestion, great care being taken, especially in hot weather, not to overfeed either in quantity of food taken at a feeding or in the number of calories given in twenty-four hours. As the hot weather approaches and the heat increases, the normal infant, thriving on a wholesome food formula, should be let alone, no attempt being made to increase the strength of the formula or to add new foods while the infant is battling with the depressing effects of the heat. Be satisfied with having a well baby during the two or three months of hot weather even if it gains little or nothing in weight. It is wise to discard the scales during this period, lest the ambitious mother attempt, by the addition or increase of foods, to maintain in the infant the same increase in weight which it was making under more favorable conditions.

Malnourished, delicate infants with feeble digestive powers are ofttimes not able to take the same amount of food during the summer months which they have previously thrived upon. With such infants it is wise, therefore, as the hot weather approaches, to slightly reduce the amount of *fat* in the food and the quantity of food at each feeding. This precautionary measure may prevent indigestion, subsequent infection, and gastrointestinal catarrh, and the infant may remain well even though it fails to gain in weight. All infants, suffering from rickets and other malnutritions, as a result of feeding with condensed milk and the proprietary foods, should during the winter months be placed upon a proper milk formula, so that their intestinal digestions may be gradually strengthened and educated to the digestion of a more wholesome food, which will gradually overcome their malnutrition and increase their powers of resistance. These measures will better prepare them to withstand the depressing effects of hot weather and perhaps enable them to resist the infection to which all are more or less exposed. The importance of this line of treatment is recognized, since feeble, malnourished infants have much less chance for life when they are attacked by these diseases.

In the prevention of the intestinal disorders of infancy the physician should recognize the fact that clean, wholesome food is the most important means for accomplishing this end. The basis of all artificially prepared infant foods should be clean, raw milk. If this be not possible, pasteurized milk, and, where the conditions are such that this is not available, then

sterilized milk. Among the very poor in our large cities it may be necessary to use condensed milk or the proprietary milk-foods, such as malted milk or Nestlé's food, for a few months during the summer; the physician deciding in the individual case that it is better to expose the infant to the dangers of rickets and other malnutritions which result from the continuous use of these foods, rather than to expose it to the greater danger from gastroenteric infections, which will almost certainly result if the infant is fed on grocery milk or other cheap grades of milk sold in the tenement districts. These milks, greatly contaminated with microorganisms, cared for without ice, and handled under unhygienic surroundings, expose the infant to dangers from gastrointestinal diseases beside which the malnutritions coming from ill-balanced, patent, sterile foods are of little consequence. The use of condensed milk and the proprietary foods may, therefore, be a life-saving measure among the very poor of our cities during the months of summer. But if one decides to give an infant rickets and other malnutritions in order to save its life, he should also feel the responsibility of curing the infant of these malnutritions as soon as the weather conditions will permit the giving of codliver oil and a return to a wholesome milk formula. The necessity for the use of condensed milk and the proprietary foods among the poor might be greatly diminished if, as Kerley suggests, tenement house mothers were furnished with sterilized milk and ice to preserve it, and if at the same time they could be systematically educated in the care and feeding of their infants. Dr. Wm. H. Park, of the New York Health Department, during the summer of 1902, demonstrated that this plan was altogether feasible. He selected fifty tenement children under one year of age, furnished them with sterilized milk and ice, placed them under the supervision of physicians who instructed the mothers in the care of the milk and the feeding-bottles, and gave the infants necessary treatment when they were ill, and as a result all of these infants passed through the summer in safety. If our municipal authorities and organized charities would undertake this same kind of work the infant mortality from intestinal diseases during the summer months would be materially diminished.

As heat is so potent a factor in producing the intestinal disorders of infancy it follows that all infants during the hot summer months should be kept as cool as possible. Among the more prosperous of our population, and especially those who live in cities, a radical change of climate is advisable. When the only thing to be considered is the welfare of the infant, it should be sent to some cool country place in our northern country or to the mountains or seashore. If this be not feasible it may be taken for the summer out of the city into the adjoining country, where the air will be purer and cooler and the surroundings cleaner. If this cannot be accomplished it should spend as much of the day as possible out-of-doors on porches or in shady yards and parks. In short, it should pass its time day and night *in the coolest, purest air available.*

Bathing in cool water promotes sleep, acts as a tonic and stimulates the

circulation, and is of great value in preventing gastrointestinal disturbances. During the very hot weather morning and evening baths are advisable. The clothing of the infant should be such as not to oppress it, and for those infants who are compelled to remain in the city very little clothing is needed. Too much clothing is a common cause of overheating, skin irritation, sleeplessness, and indigestion, all of which predispose the infant to serious gastrointestinal disturbances. Bare feet, legs and arms, light napkins, and the thinnest possible covering for the body are all that are necessary on very hot days; on damp and cool days slightly more clothing may be needed. The skin irritation which results from heat and heavy clothing causes the infant to be restless, irritable, sleepless, and thereby predisposes it to intestinal disorders. This condition may be corrected by clothing and bathing the body as above noted and dusting it with a powder made of equal parts of starch and oxid of zinc.

In the prevention of the intestinal disorders of infancy it is all-important that prompt attention be given to the earliest symptoms of gastric or intestinal discomfort. If infants had proper medical attention as soon as vomiting, diarrhea, or fever appeared, then most of these cases would never pass beyond the stage of simple gastric or intestinal indigestion. Mothers should be taught that on the appearance of these symptoms a cathartic should be given, all food should be stopped, and a physician should be consulted.

## CHAPTER XX

### ACUTE INTESTINAL INDIGESTION

**Pathology.**—There are no lesions in this disease beyond the temporary congestion and irritation of the mucous membrane which result from offensive material in the intestinal canal, when this remains long enough to produce a catarrhal inflammation we have passed beyond the stage of acute intestinal indigestion. The clinician must, therefore, ofttimes await the result of twenty-four hours of treatment to determine whether he has to deal with an acute intestinal indigestion or an intestinal catarrh.

**Symptomatology.**—Very commonly nausea, vomiting, gastric discomfort, and gastric pain may precede the intestinal symptoms. This is especially true if the attack be a sudden one, occurring in a normal child, produced by some notable error in diet. In feeble infants with weak digestive capacity, intestinal indigestion usually develops without preliminary gastric disturbance.

*Diarrhea* is the most common and characteristic symptom, although it is not always the first, and constipation may persist throughout the attack. Fever and nervous symptoms may mark the onset of the attack, and the physician may be in doubt as to the cause of these symptoms until the diarrhea assists in making the diagnosis. The very wise and almost universal custom of giving a cathartic to a child suffering acutely from fever

and nervous symptoms results in unloading the bowels, and from the character of these intestinal discharges the diagnosis is made. The stools are commonly alkaline in reaction, green in color, and foul in odor, but where they are markedly acid in reaction and sour in odor the diarrhea is more pronounced, the constitutional symptoms less severe, and the buttocks and adjacent parts red and irritated from the discharges. Mucus and undigested food are present and either small soft curds (fat) or large tough curds (casein) may be seen.

The height of the *fever* depends largely upon the age and nutrition of the infant; the younger and more malnourished it is the higher the fever. The severity of the exciting cause also influences the height of the fever, which may vary from 101° to 105° F. It falls, however, almost immediately after the bowels have been unloaded and remains normal under proper treatment; its duration should not exceed one or two days.

The *nervous* symptoms depend largely upon the age and nutrition of the child, as well as upon the severity of the exciting cause. A young, feeble, rachitic infant may have a convulsion from an exciting cause that could produce only sleeplessness and irritability in an older, normal infant. One may say, therefore, that the younger the infant and the more malnourished it is, the more severe will be the nervous symptoms, which suddenly subside when the intestinal canal is cleared. Following the fall in the fever and the subsidence of the nervous symptoms, the child may be prostrated and its face show the results of the acute illness of the previous day.

*Pain* is a frequent symptom. Intestinal colic is especially common in young, malnourished infants suffering from attacks of acute intestinal indigestion; it is produced, as a rule, by flatulency, and the abdomen is therefore more or less distended and tympanitic, but it may also result from irregular and excessive peristalsis. In the intervals between the attacks of colic, the infant remains quiet for a time and then the paroxysm is renewed; it cries fiercely, draws up its legs, twists its body, and gives every evidence that it is suffering great pain. It is also a notable fact that intestinal colic is a prominent feature of acute intestinal indigestion in older children; in these cases the pain may be very severe, the child screaming and doubling himself up in his paroxysm of pain, which comes and goes as it does in the young and feeble infant. In older children the diarrhea, fever, and nervous symptoms are comparatively slight and the patients are sleepless, restless, and irritable. The cathartic which carries away the offending material commonly relieves all symptoms.

**Symptom Groups.**—Czerny, Keller, Finkelstein, Langstein, Meyer and others attempt a differentiation of the symptom groups produced by indigestion from the different ingredients of milk. These groups represent "food injuries" not only to the digestive organs, but also to the metabolism of the infant, from the intake beyond the point of tolerance of the fat, carbohydrates, whey salts and proteins in its food. That is, overfeeding with fat, carbohydrates, whey salts or proteins produces a distinct symptom complex,

which is of great value from a clinical standpoint in that it enables one, in a given case, to withdraw from the food the special ingredient causing the trouble. If the symptoms indicate fat indigestion, then the fats are to be excluded from the diet and the carbohydrates and proteins continued; if, on the other hand, the symptoms indicate a sugar or a protein indigestion, the offending ingredient is to be discontinued and the food of the infant made up largely of the other two important ingredients. By this method the infant will not be unnecessarily starved over a long period of time when in fact there is only one ingredient which it cannot digest and properly metabolize. There must, however, in these, as in all cases of acute indigestion, be a period of complete rest and cleansing of the intestinal canal, and then the special formula, which the symptoms indicate will be tolerated, should be prescribed. It is an interesting fact that, following these "food injuries," which are so common in both acute and chronic intestinal indigestion, the infant's tolerance for the food ingredient which produced the injury is very greatly diminished, so that it may be many weeks or months before it is again able to digest and metabolize the same quantity of this ingredient which it took before the illness ("injury") occurred. For example, an infant that has been thriving upon a food formula which contained 3 or more per cent. of fat may, following an attack of indigestion, not be able to take more than ½ or 1 per cent. of fat; yet this infant may thrive on high percentages of carbohydrates and proteins during the many weeks it is slowly recovering its tolerance for fat (cream). This diminished tolerance, which may be so suddenly developed for the food ingredients of milk, is strongly suggestive of the phenomena of anaphylaxis and is perhaps similar in its pathology to many of the so-called food idiosyncrasies with which we have long been familiar. The poisoning which follows the taking of certain foods (such as eggs, fish, etc.) in certain children, and even in adults, is perhaps due to an inherited or acquired lack of systemic tolerance for these particular foods. Whatever be the explanation, the fact remains that intolerance for the various food ingredients of milk is very commonly associated with the intestinal disorders of infancy and is one of the important factors in aggravating and prolonging them. The symptom groups, however, associated with the intolerance of fat, carbohydrates, whey salts and proteins are not always clearly defined; this is in part due to the fact that different degrees of intolerance for the various food ingredients of milk may occur in the same infant, and then again it appears that in certain infants the carbohydrates may be tolerated when the food has a low fat content, or the fats may be tolerated when the food has a low carbohydrate content. The proteins, especially the casein, apparently do not aggravate the intolerance of the infant for either fats or carbohydrates, but protein intolerance is aggravated by fat and alleviated by carbohydrates. The above discussion will, the author hopes, materially assist in determining the practical value of the following syndromes.

Fat Indigestion.—Fat indigestion is very common in the gastrointestinal disorders of infancy, and apart from the ordinary symptoms of

indigestion and intoxication above noted, it has a more or less distinct symptom group. There is commonly a history of excessive quantities of fat (cream) taken. The infant is malnourished, has a pale, muddy complexion with dark circles under the eyes, has a coated tongue and fetid breath. Gastric disturbance and vomiting are common, constipation is nearly always present, but it sometimes alternates with diarrhea. The stools are, as a rule, small, fragmentary, dry, and crumbly, and are either white or light yellow in color. They may have a shiny, oily look and the odor of butyric acid may be noted. A microscopic examination will reveal an excess of neutral fats, fatty acids, and soaps. The urine may have an ammoniacal odor. An inability to digest and assimilate fat is most commonly associated with acute and chronic intestinal indigestion, the latter resulting in marasmus or atrophy.

Sugar Indigestion.—Sugar indigestion is very commonly associated with the gastrointestinal disorders of infancy; in some instances it is the important exciting cause, in others it is a contributing factor which aggravates and prolongs the disease. In these cases there is usually a history of an excessive intake of sugars (sweets); in young infants milk-sugar is the common cause. Diarrhea is the most notable symptom; the stools are frequent, copious, watery, acid, have a sour odor, and irritate the skin of the buttocks; they are usually light green in color and may contain neither mucus nor curds; gas formation, producing tympanites, is usually present. Vomiting and regurgitation of sour material are common symptoms. The infant loses weight, is irritable and fretful, urticaria may occur, and fever may be present. The urine may contain lactose, and acetone and diacetic acid are frequently found. In older children a sugar intoxication is very commonly manifested by an attack of urticaria, recurrent vomiting, migraine, or asthma, and in such cases the finding of acetone and diacetic acid in the urine strongly favors this diagnosis. In severe forms of sugar intoxication, described by Finkelstein and others, fever, profound nervous symptoms, and a well-marked polymorphonuclear leukocytosis are present. In these cases sugar intoxication is commonly associated with an inability to metabolize the whey salts, which are believed to be in part responsible for the rise in temperature. This group comprehends many of the cases which develop into severe intestinal toxemia, the symptom group of which is controlled by eliminating the sugars and whey salts from the food, and is again aggravated by the addition of these same ingredients.

Protein Indigestion.—Protein indigestion is not as common as it was thought to be a few years ago; it is, however, not infrequent. The disturbance is characterized by the ordinary signs of intestinal indigestion previously noted. There is commonly a history of an excessive intake of proteins, the infant loses in weight, is flabby, anemic, and may have either constipation or diarrhea. The stools, however, always contain large, tough casein curds; in some cases not more than one or two of these large curds are passed in twenty-four hours. The stools are usually alkaline and foul smelling. Casein indigestion very commonly leads up to a casein putre-

faction and then we may have the fever, diarrhea, and severe nervous symptoms sometimes associated with this form of intoxication.

**Diagnosis.**—The diagnosis of acute intestinal indigestion from acute intestinal intoxication and intestinal catarrh is determined by the results of the treatment. If the constitutional symptoms yield at once to proper cathartic medication and proper diet, the diagnosis of intestinal indigestion is confirmed.

The *prognosis* is good. In feeble rachitic infants, however, convulsions may occur and jeopardize life. When neglected or improperly treated this condition may be the cause of an intestinal catarrh or may excite an intestinal toxemia, either of which conditions may place the child's life in jeopardy.

**Treatment.**—From what has been said it is evident that in feeble, malnourished children who are markedly predisposed to this disease the prophylactic treatment is most important. With this type of child the diet should be closely watched and regulated to suit existing conditions. The food formula should be modified with great care to meet the needs of the individual infant, and as the summer approaches special care should be exercised to protect it from the depressing effects of the heat. If it be a city child it should, if possible, be sent into the country or placed under the best possible climatic conditions.

In dealing with an individual attack a cathartic should be given, and of all cathartics castor-oil is preferable. Even if the infant has a disturbed stomach and the castor-oil provokes vomiting, the emptying of the stomach will be beneficial, and in this event as soon as the vomiting has subsided calomel may be given. It is preferable to give the calomel in small doses, at frequent intervals, until a grain or a grain and a half has been given, and an hour or two after the last dose of calomel a second dose of castor-oil may be given. If this is again vomited, milk of magnesia in teaspoonful doses, at three or four-hour intervals, may be given until the intestinal canal is cleared. If, however, the preliminary dose of castor-oil is retained no other cathartic is needed. An enema should be given even before the cathartic; this is especially indicated if the infant is suffering from colic. The injection of water into the colon will, as a rule, unload the lower bowel and, in the great majority of instances, relieve the intestinal pain by causing a discharge of gas. The enema and cathartic having been given, the child is kept as quiet as possible, and if it is still suffering it may have dry heat applied to its abdomen; this may be done with a hot-water bottle, the hops bag, or hot flannel. If the feet be cold they also may be warmed by the application of dry heat. The cardinal rule in the treatment of these cases is absolute rest to the stomach for twelve or twenty-four hours or at least until the cathartic has thoroughly acted. Fortunately, in the beginning the child has no appetite and the thirst which accompanies the fever may be satisfied by giving small quantities of water. It is better, however, not to begin even the water until two or three hours after the preliminary cathartic. The child can, as a rule, be kept upon water for twelve or

twenty-four hours and after this, for the next day, it may be given barley water, meat juice, or thin beef or mutton broth. On the third day the diet may be whey or fat-free milk in small quantities; the milk should be largely diluted, with dextrinized barley water, and the quantity gradually increased as the child convalesces. In these cases the tolerance for carbohydrates and proteins is not reduced, and for this reason most of these cases do well upon a mixture of skimmed milk and a thick dextrinized gruel until they can gradually be returned to their original food formulas. If the symptoms indicate some definite form of food intolerance, a food formula very weak in fat, sugars or protein, as the individual case may require, may be prescribed. In severe cases the infant may have to be underfed for a number of weeks before it is finally placed upon a food formula suited to its age and weight.

There is little to add in the way of medicinal treatment for these cases. A diastase such as liquid takadiastase may be added to the barley water, and simple chalk mixture may be given if gastric or intestinal irritation continues. On the fourth or fifth day it is advisable to give a second dose of castor oil, and if this does not produce mucous discharges the child may be considered as fairly convalescent. If, however, mucous discharges are produced by the castor oil, careful dietetic treatment should be continued for a few days longer and a third dose of oil given; these cases are bordering on acute intestinal intoxication or catarrh. It is rarely necessary to use bismuth and never necessary to use opium for the control of the diarrhea in acute intestinal indigestion in very young children. The general hygiene of the nursery should be carefully looked to. The infant should have all the *fresh air* it can get without unnecessary exposure, as this is an important factor in promoting and restoring good digestion.

In *older children* it is necessary not only to give the enema and the cathartic and to insist upon abstinence from food, but it is very commonly necessary to use opium for the relief of the intestinal pain. This may be given in the form of paregoric, and if it be vomited and the intestinal colic be severe, a small dose of morphin suitable to the age of the child may be given hypodermically. In those cases where opium is used a saline cathartic is preferable to the castor oil; sulphate of magnesia will act quickly and painlessly in clearing the intestinal canal. After a period of rest to the stomach the child may be given broth, toast, malted milk, or some such simple food for twelve or twenty-four hours and then gradually return to his normal diet. During the attack hot applications to the stomach are indicated, even more than they are in the infant, and diastase or a pepsin and hydrochloric acid mixture given after meals may hasten convalescence.

## CHAPTER XXI

### ENTERIC INFECTION

(*Enterocolitis*)

Enteric infection is a broad term covering the great majority of the intestinal disorders of infancy, whose etiology and preventive treatment have just been considered. Under this condition may be grouped all the intestinal toxemias and intestinal catarrhs.

**Pathology.**—The underlying pathological condition is an infection of the intestinal canal with microorganisms, producing abnormal fermentations

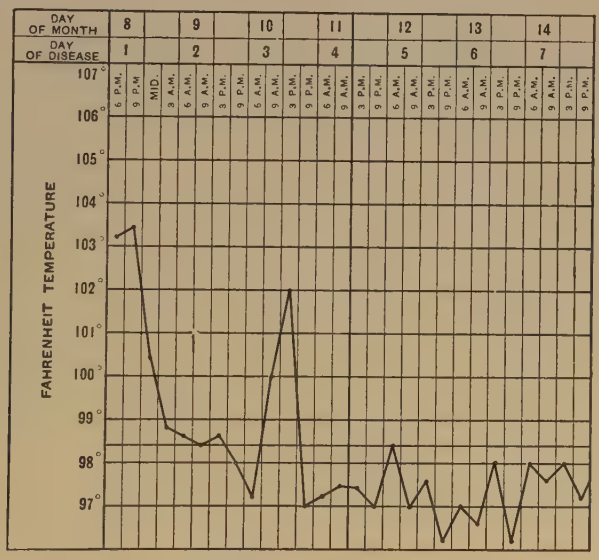



FIG. 28.—GASTROENTERIC INFECTION—MILD.

of its food contents, which result in the formation of soluble and irritant poisons. The soluble poisons are absorbed, producing a more or less severe systemic intoxication. The irritant poisons produce a congestion and irritation of the intestinal mucosa, which prepare the way for the microorganisms to produce more or less destructive lesions of the mucous membrane. In the milder cases the mucous membrane is congested, covered with mucus, and shows a beginning infiltration with round cells and also a slight loss of superficial epithelium. Peyer's patches and the adjacent nodes are swollen. As the disease progresses there develops a catarrhal inflammation of the mucous membrane of the intestine. The solitary follicles are congested and may mark the site of small necrotic ulcers, and around them superficial ulcers may spread, coalesce, and cover a large portion of the mucous membrane. In some instances this process is more necrotic, the solitary

follicles breaking down, with the formation of deep, ragged ulcers. These are especially located in the colon. The mucous membrane of the colon and lower ileum may be covered with a grayish, pseudo-membranous exudate. The spleen and liver may be enlarged, the kidneys may show degenerative changes, and the lungs may be congested and show patches of bronchopneumonia.

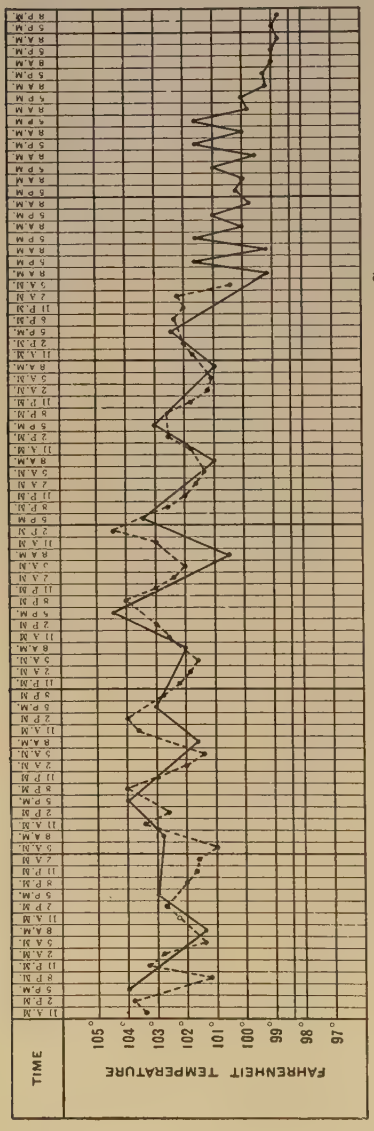



FIG. 29.—TEMPERATURE CHART OF GASTROENTERIC INFECTION—SEVERE.

**Symptomatology.**—The symptoms of this condition fall naturally in two groups, namely, those resulting from a toxemia produced by soluble toxins, and those produced by the intestinal irritation and resulting intestinal catarrh. These symptom groups, as a rule, are inseparably connected, but in most cases we have one or the other so predominating that one has little difficulty in determining whether the toxemia or the intestinal catarrh is, from the standpoint of immediate treatment, more important. The toxic symptoms are commonly more pronounced at the onset, and later the symptoms of intestinal catarrh or enterocolitis predominate. But throughout the course of this disease, in most instances, the toxic and catarrhal symptoms are so intermingled that they form a clinical picture which practically needs not be further subdivided into syndromes.

This disease is commonly marked by fever, nervous symptoms, vomiting, diarrhea, and more or less prostration, and it may vary in gravity from rapidly fatal cases of so-called cholera infantum, where the toxemia is so violent that catarrhal lesions have hardly time to develop, to mild infections showing but slight fever and nervous symptoms accompanied by a slight diarrhea.

The *fever,* which is one of the earliest symptoms, varies with the severity of the infection; it may rise within the first twenty-four hours to 104° or 105° F. It commonly continues, except in the mildest cases, for four or five days, and during this time it is markedly influenced by the action of

cathartics. As the intestine is unloaded of its poisonous contents the temperature falls sharply, perhaps rising again to be again lowered by cathartic medication. If the fever continues beyond the fifth or sixth day one may conclude that catarrhal lesions in the intestine have formed, and the disease from this time on presents more marked symptoms of enterocolitis and, in favorable cases, less marked symptoms of toxemia. The persistence or return of well-marked toxic symptoms in these cases is a very unfavorable indication.

*Vomiting* is usually the symptom which calls attention to the child's illness. It occurs in a large percentage of the cases and may be most distressing and troublesome during the first twenty-four or forty-eight hours. Within this time under appropriate treatment the vomiting disappears and does not commonly return, but when it does return it is an unfavorable indication, as it generally means an increase in the intestinal toxemia. The *diarrhea* which occurs in this disease is, like the vomiting, an effort on the part of nature to get rid of the poisons in the gastrointestinal tract. The earlier the diarrhea appears the better for the patient, as it reduces the fever and other toxic symptoms. This eliminative diarrhea usually begins on the second day. The fecal discharges vary greatly in different cases; they are commonly foul in odor, discharged with flatus, green in color, and contain mucus and undigested food. The number of discharges may vary with their size; in some instances being small and frequent, in others copious and discharged at intervals of four or five hours. In this disease, above all others, it is important to bear in mind that the diarrhea, especially during the first few days, is a life-saving measure, which is to be encouraged by proper medication. If constipation be present, as it is in some cases, then the vomiting and constitutional symptoms are aggravated and the necessity for unloading the bowel more urgent. As the catarrhal process becomes more marked in the colon and in the lower ileum the intestinal discharges are then made up largely of bloody mucus, which is passed with more or less pain and straining and the number of stools may be as high as twenty or thirty in the twenty-four hours. This character of stool occurring in a child acutely ill and having an elevation of temperature, whether it occurs early or late in the disease, indicates that the infection has produced an enterocolitis. It should, however, be noted that blood in the stools does not always mean intestinal ulceration; if transitory in character it commonly means a simple catarrhal process with marked congestion. On the other hand, it should be noted that marked ulceration may occur without blood showing in the stool at any time. The diagnosis of ulceration of the bowels, however, may be assumed if the fever and mucopurulent discharges, tinged or not with blood, continue for ten days or two weeks. Prolapse of the rectum is not uncommon in these cases and in rare instances a pseudo-membrane may be seen on the prolapsed rectum. Small pieces of pseudo-membrane may also be found in the stool. These appearances are the only positive indications of the presence of a pseudo-membrane in this disease.

The *nervous symptoms* are most important and their severity will depend upon the virulence and amount of soluble toxins absorbed from the intestine, and upon the susceptibility of the individual infant to the action of these poisons. Restlessness, fretfulness, muscular twitchings, somnolence, stupor, convulsions, delirium, unconsciousness, and finally paralysis of vital centers resulting in death may occur. In severe cases a peculiar nervous syndrome closely resembling meningitis may be present. Here the stupor and convulsions may be associated with retraction of the head, stiffening of the muscles of the neck, irregular pulse and respirations. These symptoms continuing for days, with fever and increasing coma, may be distinguished from meningitis by the fact that they are associated with the character of diarrhea previously described, and by the absence of characteristic findings in the cerebrospinal fluid obtained by tapping the spinal canal. Since this nervous syndrome is not associated with meningeal lesions it is perhaps produced by the action of toxins on the nerve centers. In severe cases emaciation is extreme, the eyes are sunken, the fontanels depressed, and the infant may die from exhaustion or pass into an atrophic condition from which it requires months, if not years, to recover. Fortunately, however, the majority of the cases of intestinal infection are of moderate severity and associated with a mild form of toxemia. In these cases the nervous irritability, muscular twitchings, mental stupor and fever gradually subside. The blood, if present, disappears from the stool, but the mucous discharges continue with more or less tenesmus for six or seven days, with gradual improvement until they become normal at the end of the second or third week.

Urticaria, erythema, and other toxic rashes are very common, especially in the milder cases.

The general appearance of the infant is an indication difficult to describe, but one that impresses the physician almost more than any other with the seriousness of this disease. The character of the prostration, the facial expression, the extent of the emaciation and the mental alertness of the infant vary in the production of a picture, which greatly assists the physician in making his prognosis in individual cases.

Cholera infantum represents the most severe type of acute enteric infection. It has no distinguishing characteristics except the suddenness of its onset and the severity of its symptoms. This clinical syndrome resembles in some particulars that of true cholera, and it has, therefore, been dignified by the title cholera infantum. It usually occurs in non-resisting weaklings, but may occur in normal infants. It is believed to be a food poisoning commonly produced by badly contaminated milk; the infant gets such a large initial dose of toxins that it is almost immediately overwhelmed by the toxemia. The choleriform diarrhea produces more rapid emaciation and loss of weight than any other disease of infancy. The stools are large, frequent, and watery in character and this violent purging is accompanied by severe vomiting. The temperature commonly reaches 104° or 105° F., continues high

for twelve or twenty-four hours, and then falls with the collapse and prostration of the infant. The surface temperature is cold, while the rectal temperature, in the rapidly fatal cases, may register 107° F. Stupor, convulsions, and coma, following each other in rapid succession, may mark the progress of the disease, which may terminate fatally within twelve hours, but which commonly lasts for three or four days. Under proper treatment some of these cases, especially those occurring in previously normal children, may recover.

**Prognosis.**—Other things being equal, the younger the infant the more unfavorable the prognosis. The prognosis also depends upon the severity of the enteric infection, the resistance of the individual infant, the hygienic surroundings, and the character of the treatment instituted. On the whole, however, except in cholera infantum, the prognosis is good, since the vast majority of these cases are produced by infections so mild that they are readily controlled by appropriate treatment. The continuous presence of bloody mucus in the stools for weeks indicates ulceration in the bowels, which means either an unfavorable prognosis or a very slow recovery.

**Treatment.**—As this condition in the beginning is essentially an acute intestinal poisoning, the all-important indication (except in those cases of the cholera-infantum type) is to cleanse the intestinal canal as rapidly as possible. With this end in view the colon should be irrigated and immediately thereafter a dose of castor-oil or sulphate of magnesia given; the oil is to be preferred unless the stomach be very irritable. If the oil or magnesia is rejected by the stomach, it should be allowed to rest for half or three-quarters of an hour and then washed out with a solution of sodium bicarbonate, a teaspoonful to the quart. Half an hour following this irrigation calomel should be administered, one-quarter of a grain combined with one grain of sodium bicarbonate, every half-hour, until two grains have been given. After the preliminary cathartic has cleared the intestinal canal there is commonly no further vomiting and all the toxic symptoms, including the fever, are much modified in their severity, but in the event that the vomiting continues to be a troublesome symptom, the stomach may be washed out with normal salt solution every six to twelve hours for the first twenty-four or thirty-six hours. Lukewarm baths, sponging with cool water, and ice caps to the head are valuable measures in the control of fever and nervous symptoms during this stage of the treatment.

Diet.—It is evident that in a disease where the unloading and cleansing of the intestinal canal are of such vital importance, it must be equally important that during the first few days little or no food should be given. During the first twenty-four hours only water should be given; during the second twenty-four hours weak tea is an excellent substitute for food. Alcohol in the form of good whiskey or brandy may also be given in from 15- to 30-drop doses, well diluted, every three or four hours, provided the stomach is in a condition to retain it. During the third twenty-four hours, if the infant be breast-fed, it may be allowed to nurse

at intervals of five or six hours, until it has been demonstrated that the breast milk will not cause a return of the symptoms, and within the next few days it may have the normal quantity of breast milk. In artificially fed infants, which make the great majority of the cases, the infant may on the third day be given barley water, toast water, or a weak lamb or beef broth. In very young infants the barley water should be dextrinized, as undigested starch is not easily assimilated at this age. By the fourth day, if the case has progressed favorably and the fever and other toxic symptoms have subsided, one part of skimmed milk mixed with four parts of dextrinized barley water may be given. If this agrees, the skimmed milk and beef or lamb broth may gradually be increased in quantity until convalescence is thoroughly established, and then the infant should be gradually returned to its original diet. In more severe cases, after the acute symptoms of the disease have been controlled, and the infant still has unhealed ulcers in its intestinal canal and a feeble digestive capacity, the skill of the physician will be taxed to give it food which will sustain it and at the same time will not cause relapse. Breast milk is the safest and best of all foods to accomplish this result. If a wet-nurse cannot be obtained Finkelstein's albumin milk, or the curd from a pint of cow's milk which has been finely disintegrated by passing it through a sieve two or three times, may be added to a pint of equal parts of skimmed milk and barley water, and one or the other of these foods be given in proper quantities until the symptoms of intoxication and intestinal irritation have subsided. Fresh buttermilk, and the buttermilk and malt soup formulas elsewhere described are useful in some cases.

The principles of importance in the dietetic management of these cases are:

*First.*—Starvation for a variable length of time, depending upon the severity of the case; during this time albumin water and beef juice, so commonly recommended, are not to be given. In fact, these albuminous foods are especially contraindicated at this time, and continue to be until the putrefactive processes in the intestine have been controlled.

*Second.*—Breast milk is by far the best food in these cases, but early in the disease it should be given in small quantities and at long intervals. If artificial food must be used it should be poor in milk and cane sugars and whey salts and rich in casein. Weak carbohydrates, such as barley water, dextrinized gruels, and dextri-maltose foods may be used to dilute the casein mixtures. These foods may be inoculated with lactic acid bacilli so that they may act as the breast milk does by *increasing the normal intestinal flora,* which antagonize the pathogenic flora which have taken possession of the intestinal canal. Fresh buttermilk acts in the same way.

*Third.*—As the convalescence of the infant is established, the food is to be very gradually increased by the substitution of ordinary milk formulas. There is great danger of reinfecting the intestinal canal by overtaxing it, especially with whole milk, lactose, saccharose and whey salts.

*Fourth.*—In more severe cases, where the disease is prolonged by catar-

rhal or ulcerative processes in the colon and lower ileum, the dietetic treatment is much more prolonged and difficult, and a wet-nurse is not only advisable but absolutely necessary to successful convalescence. If a satisfactory wet-nurse cannot be obtained then a very slow return to the original diet will be necessary, and this period may extend not only over weeks but over months.

Medicinal Treatment.—Other medical treatment than that above given for cleansing the intestinal canal may be of value in individual cases, but it should not interfere with the preliminary cathartic medication and should not, as a rule, be begun until the second or third day. To allay the gastric and intestinal irritation 3- to 5-grain doses of equal parts of compound chalk powder and guaiacol carbonate may be given every three or four hours, and a day or two later for the relief of the same symptoms, one may substitute for the above prescription 1 or 2 grains of salol and 3 to 6 grains of subnitrate of bismuth given in one-half-teaspoonful doses of simple chalk mixture. If the diarrhea persists and the disease becomes less acute, the doses of bismuth may be increased to 10 grains every four hours. Diastase aids the digestion of carbohydrate foods, and hydrochloric acid and pepsin, taken after eating, assist in the digestion of milk and other protein foods and are of value during convalescence.

Morphin in 1-50 to 1-100-grain doses is a remedy of great value for controlling convulsions where simpler measures fail, while the fact should be impressed that morphin given in this way may be a life-saving measure, yet it should also be noted that the practice of giving morphin for the control of convulsive disorders may be abused and may result in harm to the infant. In the severe cases, however, where the convulsions are continuous, this is practically the only remedy that can be relied upon. Holt recommends 1-50 grain of morphin and 1-600 grain of atropin, to be given hypodermically, to "neutralize the effect of poisons on the heart and nervous system in cases of the cholera infantum type." This use of morphin is almost the only indication for the opium preparations in the treatment of acute gastroenteric infections; it is perhaps never necessary to give opium to *infants* under one year of age for the control of the diarrhea or for the relief of pain or other intestinal symptoms; the indiscriminate use of paregoric and other opium preparations given by the mouth to relieve pain and diarrhea, increases the mortality in this disease. In older children, after the acute symptoms have subsided, the opium preparations, especially paregoric and Dover's powder, may be of value in relieving the pain and tenesmus which occur when the catarrhal condition becomes localized in the colon. But even in these cases they must be used with great discrimination, if good rather than harm is to result from their use. Colon irrigations are of special value in those cases which, as shown by the large quantities of mucus they pass, have catarrh or ulceration of the colon, and, as the object of these irrigations is largely to remove the mucus and otherwise cleanse the diseased mucous membrane without irritating it, there can be little doubt that lukewarm normal salt solution will serve this purpose best.

Irrigations of a sufficient amount of this fluid to fill the colon may be given from one to three times in twenty-four hours. If the infant does not respond kindly to these injections or shows prostration or intestinal irritation following their use, they may be discontinued. Such unfavorable symptoms, however, are more commonly due to faults of technique than to the injections themselves, and there can be no doubt but that on the whole they are a very valuable aid in the treatment of this disease. In the event that catarrh or ulceration of the colon continues for weeks after acute constitutional symptoms have disappeared, good may then result from astringent injections of alum or tannic acid (one drachm to the quart of water), following the cleansing of the membrane with normal salt solution. Castor-oil is the cathartic par excellence, not only in the beginning but throughout the course of this disease. It is especially valuable in those cases characterized by the passage of frequent, small, mucous stools; not only in these cases, but in all it is to be repeated every third or fourth day unless it be contraindicated by the condition of the stomach or some idiosyncrasy on the part of the child. No other laxative acts so kindly in carrying off the mucus and relieving the complicating toxemia as does castor-oil. If some other laxative must be used, then Rochelle salts, the milk or sulphate of magnesia may answer the purpose.

Stimulating Treatment.—Stimulating treatment by hypodermic medication may be a life-saving measure in very severe cases. If, for example, during the first few days, when it may be impossible to give stimulants by the mouth, the infant should be threatened with symptoms of collapse, hypodermic stimulation is not only advisable but is ofttimes absolutely necessary. The most valuable stimulant we possess is normal salt solution (45 grains of sodium chlorid to 1 pint of sterile water). This should be given by hypodermoclysis, 6 to 8 ozs. at six-hour intervals. The salt solution is rapidly absorbed, acts as a general stimulant, furnishes fluid to the body media, and assists in the elimination through the kidneys of the absorbed toxins which produce such a profound influence upon the nervous system. Caffein sodium benzoate in ¼ to ½-grain doses may be given hypodermically. Camphor and ether are valuable stimulants and may be used as directed in the following prescription modified from Forchheimer:

℞ Camphoræ ..... 25 grs.
Olei amygdalæ express ..... ½ oz.
M. Five to ten minims hypodermically.
℞ Camphoræ ..... 25 grs.
Æther ..... 2 drachms
Olei amygdalæ express..... 2 drachms
M. 5 to 10 minims hypodermically.

The above hypodermic medication is especially valuable in the treatment of cases of cholera infantum. By these measures the life of the infant may sometimes be prolonged until the vomiting and diarrhea subside, permitting the administration of water and stimulants by the mouth.

Hygienic Treatment.—The hygienic treatment of these cases is of the utmost importance. While acute symptoms threaten the life of the child it should be kept *at home* and there placed under the most favorable hygienic conditions possible. Many infants are rushed away from their homes when acutely ill with enterocolitis, with the idea that cool, pure country air is of more importance than skilful medical attention and careful nursing in the treatment of this disease. Many such infants subjected to long railroad journeys die on the road or soon after they have reached this "ideal climate," where perhaps they have not found good medical attention and skilful nursing. *Home* is the place to begin the treatment of acute enterocolitis and the treatment should there be continued until the acute symptoms are under control and the infant is in a condition to travel without danger of a relapse; then it should be sent for its convalescence to some bracing climate where it can have pure cool air.

## CHAPTER XXII

### CHRONIC INTESTINAL INDIGESTION

(*Chronic Enterocolitis, Infantile Atrophy, Marasmus*)

### CHRONIC INTESTINAL INDIGESTION IN INFANTS

**Etiology.**—The general etiology of this condition has been previously outlined; here, however, certain factors prominent in the chronic forms of intestinal diseases are emphasized. Age is an important predisposing factor; most of these cases occur in infancy, but they are not uncommon in the child. Among predisposing causes the following may be noted: Heredity, rickets, anemia, tuberculosis, syphilis, general malnutrition, and previous intestinal attacks. The infant may inherit from weak and neurotic parents a physiological incapacity to digest ordinary food. The heat of summer, impure air, and dirty surroundings are most potent factors in producing chronic gastrointestinal disturbances.

The exciting causes are to be found in the food. If the disease occurs in breast-fed babies it is due to overfeeding, too frequent feedings, or to some fault in the breast milk. Improper artificial feeding is, however, the cause in the vast majority of cases. Too much food, in ounces given at a feeding and in the number of calories in twenty-four hours, overtaxes the digestive powers of the infant and produces repeated attacks of acute indigestion and intoxication, which in time so diminish its normal digestive capacity that it is in a more or less constant state of intestinal indigestion. The infant, after repeated attacks of acute intestinal indigestion, very commonly has its digestive capacity for fat and whey salts very much lowered. Wholesome food, therefore, given in too great quantities or containing too many calories or given at irregular intervals, may, after a time, produce chronic intestinal indigestion. Food spoiled by bacterial contami-

nation is the most potent cause of this condition; it acts by causing repeated attacks of acute intestinal infection which in time produce chronic indigestion.

**Pathology.**—The underlying condition is a pathological digestive incapacity which makes it impossible for the infant and child to digest, assimilate, or metabolize the ordinary food suitable to its age and weight. The degree of this incapacity determines the seriousness of the condition. In many cases there is present a low grade of intestinal catarrh, so that these cases might properly be classed as *chronic enterocolitis*. But from a clinical standpoint this differentiation is not important since the treatment is the same.

**Symptomatology.**—Profound increasing *malnutrition* associated with a *chronic diarrhea* is the most characteristic symptom. The number of discharges in the diarrheal cases may be many or few in a day; they contain mucus, curds and undigested food, are commonly green in color, and may be either foul or sour in odor; they are passed with flatulence, but there is little pain. The buttocks may be irritated or excoriated from the discharges. The stools usually contain neutral fats, fatty acids and soaps, and the curds are commonly small and soft; tough, large, casein curds holding in their meshes large quantities of fat are also frequently seen. Those cases in which the diarrhea is a marked symptom are commonly due to an incapacity to digest sugars and fats. In other cases constipation may be a marked symptom and it may alternate with diarrhea. In the constipated cases the stools are commonly gray or white in color, of a putty-like consistency, covered with mucus, and have a foul odor; there is usually more or less fever, colic, and nervous symptoms. No hard and fast lines can be drawn separating the diarrheal from the constipated cases, either in their symptomatology or their etiology, since in the same infant the two symptom groups not uncommonly alternate; but the dry, putty-like, putrid, constipated stool is commonly associated with fat indigestion, and the copious, watery, frequent, diarrheal stools, sour in odor, discharged with flatus and irritating to the buttocks, are usually associated with sugar indiges-

Fig. 30.—Casein Curds, Actual Size. (Talbot.)

tion. Protein indigestion may be associated with either of these symptom groups. *Fever* is commonly absent; subnormal temperatures are common. Fever, however, associated with other acute symptoms, is an indication that an intestinal infection, or an acute intestinal indigestion, is complicating the chronic process; this not infrequently occurs during the prolonged course of a chronic intestinal indigestion. *Nervous symptoms* are more marked in the constipated cases. The *appetite* is usually good, the infant very commonly empties its bottle and cries for more; this appetite far beyond its digestive capacity is a source of great trouble, since the mother and sympathetic relatives are led to believe that the infant is being starved, and, as its appearance carries out this inference, it is often difficult to feed it within the range of its digestive capacity. The urine usually contains indican or indolacetic acid and acetone may be present.

FIG. 31.—INFANTILE ATROPHY.

INFANTILE ATROPHY. —The common causes of this condition are prematurity, chronic tuberculosis, syphilis, chronic intestinal indigestion, chronic enterocolitis, improper food, faulty methods of feeding or an hereditary physiological incapacity of the digestive organs. It is largely an institutional disease, occurring especially in artificially fed children. Wentworth believes it is due to "a defective correlation of the several digestive organs, stomach, intestines, pancreas and liver," which results in disturbed digestion and feeble assimilation of foodstuffs. Edsal has demonstrated the absence of digestive ferments in some of these cases. Finkelstein and other German writers believe that it is largely due to an inability of the infant to digest and assimilate fat (cream), and the whey salts.

The symptomatology in its early history is commonly that of intestinal indigestion. With the progress of this disease the infant loses in weight, becomes anemic, malnourished, rachitic, and finally profoundly emaciated.

The picture presented by advanced atrophy is truly a pitiful one; the face is thin, wistful, senile, the eyes are sunken in their sockets, and the fontanels depressed; the neck, arms, and legs are atrophied; the subcutaneous fatty tissue having disappeared, the dry skin hangs in loose folds over the bony structures and a general edema may be present. The abdomen is distended and tympanitic, and the bones of the chest and back stand out prominently beneath the skin which enfolds them. In these advanced cases the stools may be apparently normal, but a microscopical examination, as a rule, shows undigested food, with an excess of neutral fats, fatty acids and soaps.

**Prognosis.**—The prognosis of intestinal indigestion depends upon the length of time the disease has lasted, the degree of malnutrition it has produced and, above all, on the amount of injury which has resulted to the infant's digestive and assimilative capacity. In severe cases which have reached the stage of "atrophy" the prognosis is very grave, in the milder cases there is a fair chance of recovery; in all cases the convalescence is slow, and success largely depends upon the physician's ability to impress upon the attendants the necessity for long-continued attention to the minutest details in the care and feeding of the infant.

**Treatment.**—In beginning the treatment of chronic intestinal indigestion a dose of castor-oil should be followed by abstinence from food for a period of twelve or twenty-four hours, and the infant should then be given the following dietetic treatment:

Dietetic Treatment.—In the beginning the mother should be told that the object of the dietetic treatment is to find a food that can be digested and assimilated in small quantities, and that the evidences of success in treatment will be manifested by the improvement in and gradual return to normal of the intestinal discharges and by the infant beginning to hold its own in weight, but that under no conditions is it expected to gain in weight for some weeks to come. Such an understanding is necessary in order to have the co-operation of the mother and nurse throughout the long and tedious treatment. Human breast milk is the most valuable of all foods in the treatment of these cases and should, therefore, be used when it can be obtained; it is absolutely necessary in the worst cases, those approaching the atrophic type. In these cases of infantile atrophy it may be necessary to resort to small feedings of skimmed breast milk to give the nutritional processes a start, and then continue the dietetic treatment with small quantities of whole breast milk nursed from the breast at long intervals. The success which sometimes follows the feeding of these apparently hopeless cases of atrophy with human milk depends partly on the fact that it contains certain ferments which assist in its own digestion, and, since the fat of the milk is the most potent disturbing factor in these cases, the fat-free mother's milk is the ideal food with which to begin the feeding. The second important fact to be remembered is that whatever food is taken it must in the beginning be given in very *small quantities,* 2 to 4 ounces at four-hour intervals, and only very gradually increased when

it has been demonstrated that it has been digested and assimilated. Water may be given in the intervals between the feedings.

When breast milk is not available the infant should be placed upon a fat-free milk, peptonized for twenty or twenty-five minutes, and then placed on ice without heating. When fed it should be diluted one-half with boiled water, to which lime water has been added. After a week or ten days a dextrinized gruel may take the place of the water in the above mixture. The fat-free peptonized milk and dextrinized gruel mixture, if it agrees with the infant, should be gradually adjusted, in ounces given at a feeding and in calories given in twenty-four hours, to suit the weight of the individual infant. After some weeks, if the infant is progressing favorably, small quantities of fat may be gradually added to the food. This is best done by skimming the milk a little less closely. Slowly the fat is, in this way, returned to the food formula and then, perhaps after months of treatment, the milk is slowly depeptonized. That is to say, the peptonizing process is carried on for a shorter time and with less peptonizing powder until, in the course of a few months, it is discontinued.

Finkelstein's albumin milk, buttermilk, and malt soup formulas are of value in the treatment of many of these cases. Beef juice, whey, and albumin water may be used to supplement the diet when other foods are given at long intervals. In the convalescence from this condition the general rules previously outlined under Infant Feeding should be followed.

Hygienic.—Fresh air and a suitable climate are very necessary to success. During the hot summer months these patients should, if possible, be sent out of the city to a cool country place in the north or to the mountains or seashore, where they may live out-of-doors. These delicate, malnourished infants, when kept out-of-doors, often require artificial heat in the form of hot-water bottles to keep their bodies and extremities warm. They also require quiet surroundings so that they may have all the undisturbed sleep possible, and regular bathing for its stimulating and tonic effects.

Medicinal.—The medical treatment is of secondary importance. When fever or nervous symptoms develop a laxative such as castor-oil or milk of magnesia should be given and the following prescription used:

℞ Misturæ cretæ ..... ℥i
Liquid taka-diastase ..... ℥i
Sig. One-half to one teaspoonful with each feeding.

## CHRONIC INTESTINAL INDIGESTION IN OLDER CHILDREN

**Etiology.**—This condition is exceedingly common and is frequently overlooked, being mistaken for some functional nervous disorder. Its underlying cause is usually a carbohydrate intolerance; it is aggravated and prolonged by the excessive use of sugars and starchy foods, especially potatoes. The use of candies, sweets of all kinds and other foods unsuited to the child's digestive capacity are potent factors. Eating between meals

and at irregular intervals will aggravate and prolong the disease. It occurs very commonly between the ages of three and eight, and is more frequently seen in neurotic children suffering from other constitutional diseases.

**Symptomatology.**—These children are neurotic, malnourished, thin, anemic, and delicate in appearance. They have little powers of resistance, and, when fatigued, have dark circles under their eyes. Their appetites are poor and capricious; nausea, vomiting and fever may occur from slight causes; the tongue is coated, thick and flabby; the breath at times has an acetone odor. They are usually constipated, have large distended abdomens, with more or less marked tympanites. The intestinal discharges produced by laxatives or enemata consist of undigested fecal masses, mixed with mucus and fluid matter of foul odor. The stools may be white or dark-brown in color, with a coating of mucus as their distinctive characteristic. Following a cathartic, such as castor-oil, the mucus is passed in large amounts. The urine may contain acetone or diacetic acid, and almost always has an excess of indican or indolacetic acid. The nervous symptoms are very pronounced; they constitute, in fact, the symptom group which calls attention to the child's illness. The child is irritable, fretful, sleeps badly, dreams and cries out in its sleep, and frequently has night-terrors. Fainting spells, asthma, severe headache and even convulsions, resembling epilepsy, may occur. The following case illustrates a severe type of this condition.

Boy, age five years, had never been strong, had stomach and intestinal trouble very frequently during his life, was thin, anemic and malnourished. During the past year he had been very nervous. This nervousness increased so that he was irritable, cried on the slightest provocation, was very restless at night, and had certain peculiar nervous attacks. These attacks came on suddenly with dizziness, the boy fell to the ground, his mother thought that he did not lose consciousness, was sure that he had no convulsive movements, and some minutes elapsed before he was able to regain his feet; they were followed immediately by severe headache, more or less nausea, and a profound sleep which lasted some hours. From this sleep he awoke quite as well as before the attack. Seven of these attacks occurred during the past year, and they increased in frequency and severity. They apparently occupied the border line between migraine and epilepsy. This boy suffered more or less constantly from constipation, abdominal distention and flatulency. The constipation at times, however, gave way to an attack of diarrhea. He had no fever, and his mother said she fed him almost anything he would eat, "because he ate so little that it could not hurt him!" The urine was highly colored, had a specific gravity of 1.023, contained no albumin, no sugar, but there was a marked *excess of indican.* This boy promptly recovered under treatment.

Intestinal indigestion in the older child is frequently associated with *constipated, mucous, foul-smelling, undigested stools, and is characterized by intestinal toxemia, profound nervous symptoms, great excess of indican in the urine and progressive failure in general health.* These cases, not

associated with diarrhea and intestinal pain, are most commonly overlooked.

**Prognosis.**—This is good under proper treatment. The disease, however, is essentially a chronic one, and it should be understood in the beginning that it will require many months, perhaps years, to restore the child to perfect health.

**Treatment.**—This is chiefly DIETETIC. It is most important that the child should eat suitable food at proper intervals, taking absolutely no food between meals. From three to four meals a day should be prescribed, depending upon the age and digestive capacity of the individual child. Food should be eaten slowly, well masticated, and violent exercise directly after eating should be avoided. The following foods are especially contraindicated: cakes, candies, sweets of all kinds, an excess of starchy foods, potatoes, hot breads, fried foods, pastries, and raw fruits. In some cases, especially those in which the stools are white and fragmentary, all fats, such as butter, cream, fat meat and codliver oil, are to be excluded from the diet. The following foods may, as a rule, be recommended: beef, lamb, chicken, fish, eggs, peptonized milk, skimmed milk, malted milk, beef juice, broth, toast, a small quantity of bread, and certain well-cooked cereals, such as rice, cream of wheat, oatmeal, farina, arrowroot and tapioca. In taking these cereals as little sugar as possible should be used, and they should be covered with milk rather than with cream. As the child convalesces stewed celery, carrots, asparagus tips, peas, beans, orange juice, baked apples, and prune juice may be allowed. This diet should be persevered in for many months, and above all sweets and potatoes should be excluded until convalescence is established.

A suitable CLIMATE, in which the child may live and sleep out of doors and take exercise in the fresh air, will greatly hasten convalescence.

MEDICINES play a useful rôle in the treatment of this condition. The careful choice of laxatives is important. A dose of castor-oil is occasionally necessary, especially when acute symptoms develop. It is most important that the bowels should be moved daily. This may be accomplished by milk of magnesia, cascara, or at times by saline enemata. The injection at bedtime of one to three ounces of olive oil into the rectum, as recommended by Kerley, is very valuable. Systematic massage is also valuable in overcoming constipation. Hydrochloric acid and pepsin taken after meals are of value in some cases. The thick malt extracts containing diastase are especially useful in promoting the digestion of the carbohydrates. In some instances nux vomica and the organic iron preparations may be combined with the malt extracts, to the advantage of the patient. With the judicious use of these measures chronic intestinal indigestion in the older child can be successfully treated, provided the physician is content to go slowly with his dietetic treatment, and provided he has an ever-watchful mother or nurse to see that the details are carried out.

# CHAPTER XXIII

## CHRONIC CONSTIPATION IN INFANCY AND CONGENITAL DILATATION OF THE COLON

### CHRONIC CONSTIPATION IN INFANCY

**Etiology.**—There is on the whole a natural predisposition to constipation in infancy which is in part counteracted by mother's milk and is aggravated, as a rule, by the ordinary modified milk formulas used in artificial feeding. This predisposition lies largely in the fact, as Jacobi has so clearly pointed out, that the colon is relatively longer in the infant than it is in the adult, and that especially the sigmoid flexure runs a winding convoluted course with a long mesenteric attachment. This condition of the colon in infancy furnishes a suitable reservoir for the collection and retention of fecal matter. As the child grows older and its body increases in size, the sigmoid flexure becomes less convoluted and gradually approaches the condition found in the adult.

Rickets, anemia, and especially long-continued intestinal fermentation result in a weakening and thinning of the muscular fibers of the intestines, which interfere with normal peristalsis and lead to dilatation of both the small and large gut, thus producing a condition of muscular atony, which is a very common cause of constipation in infancy. Heredity may be an important etiological factor. Constipation is not infrequently a family disease. Functional incompetency of the liver, occurring periodically, may aggravate a mild into a very obstinate constipation. The temporary absence or deficiency of bile produces dry, putty-like, putrid movements, and this intestinal condition is associated with lack of appetite, coated tongue, bad odor of the breath, and in older children headache and nausea. Constipation is also common following the acute infectious diseases, and especially so during and following an attack of meningitis. Diseases of the rectum, such as fissure and hemorrhoids, may produce a reflex spasm of the sphincter muscles and may cause the child to resist as long as possible the desire to go to stool. Simple non-incarcerated hernia may also be a cause. Pyloric stenosis and inflammatory bands produced by peritonitis may cause serious forms of constipation. The frequent use of purgative medicines, especially castor-oil, may be a factor in converting a simple into a chronic constipation. Irregularity as to time in evacuating the bowels, which results from lack of proper training of infants and young children, and from the haste of older children to go to school in the morning and their confinement during school hours, is a cause of constipation.

Dietetic causes are more important than all other factors in producing this condition. In breast-fed babies constipation most commonly results from insufficient food and from a low percentage of fat in the milk. The mother's milk may be modified to overcome this condition by placing her

under proper hygienic conditions and regulating her diet according to the principles elsewhere outlined. In artificially fed infants the sterilization and pasteurization of milk and the giving of easily digested carbohydrate mixtures, weak in fat and proteins, are common causes of constipation. In older children the feeding of easily digested foods, such as milk, eggs, meat, cereals, white bread, toast and broths, to the exclusion of fruits and vegetables, is a potent factor in producing this condition.

**Symptomatology.**—Constipation is determined, not by the number of stools, but by the character of the discharges. The normal infant may have from one to four soft evacuations in the twenty-four hours, but when constipated the stools are composed of hard, dry, round or fragmentary fecal masses passed with difficulty and with more or less straining and tenesmus. These discharges may occur two or three times in the twenty-four hours or an interval of days may elapse between them. The fecal masses may also be covered with mucus and stained with blood. The infant loses its appetite, is restless, sleepless, nervous and irritable; it has attacks of colic with abdominal distention; it may suffer from frequent attacks of intestinal indigestion with vomiting, fever and severe nervous symptoms, even convulsions. This latter symptom group is due to the intestinal toxemias produced by the constipation and occurs especially in rachitic, malnourished infants. In these cases an increase in the indican in the urine may be a valuable indication of the onset of an intestinal toxemia.

In older children an excess of indican or indolacetic acid in the urine is a more valuable indication of the extent of the putrefaction going on in the intestine. The intestinal toxemia which results from the constipation is not infrequently the cause of a more or less profound malnutrition and anemia, especially of the chlorotic type. These children may also suffer not only from colic and indigestion, but frequently from obscure nervous symptoms such as headache of the migrainous type and attacks of "recurrent vomiting." Vertigo and temporary loss of consciousness may form the nucleus of a symptom group, which may ultimately develop into epilepsy if the constipation and resulting intestinal toxemia are not relieved.

In both infants and older children fissure and prolapse of the rectum, and hemorrhoids may result from chronic constipation and may then aggravate the condition.

**Diagnosis.**—The diagnosis of constipation, as the all-important cause of a severe nervous syndrome or of a profound anemia, may sometimes be overlooked, especially in older children. It is therefore important for the physician to keep in mind the fact that such severe symptom groups may have their origin solely in a chronic constipation. A careful examination of the abdomen may reveal in these cases impacted fecal masses in the colon. This examination should never be omitted in any case of constipation, however mild it may appear. It is also important before beginning the treatment that the cause of the constipation should be made out. The family history as to heredity should be carefully noted. The rectum should be examined for local disease and the genitalia for sources of reflex irritation;

phymosis is of special importance. These abnormalities about the rectum and genitourinary organs may locate the cause of the constipation. The form of constipation which occasionally results from an excess of cream in the infant's food should also be kept in mind; in this condition the stools are gray, dry, fragmentary and putrid. It is also most important to determine whether an atonic condition of the intestine is the cause of the constipation; in these cases the abdomen is distended with gas, the infant is anemic or rachitic, and enemata are not effective in unloading the bowels; an active cathartic, however, with temporary abstinence from food relieves the distended intestine and flattens out the abdomen.

**Treatment.**—Treatment in Infancy.—It should be understood in the beginning that constipation, especially in the artificially fed infant, is so common that one may almost consider it the normal condition, and it should also be understood that this mild form of constipation occurring in infants under one year of age cannot, as a rule, be altogether cured by diet. It must therefore be relieved by the introduction into the rectum of an oiled catheter or a gluten suppository, or a small quantity of normal salt solution or olive oil. At this age glycerin and soap suppositories should not be habitually used, as they may produce local disease of the rectum and thus aggravate and complicate the constipation. Medicines are also of value, especially the milk of magnesia, which may be used occasionally to supplement the local measures above described. Castor-oil is contraindicated unless an intestinal intoxication with fever and constitutional symptoms complicate the constipation. Diseases of the anus and rectum should be relieved by appropriate treatment.

The *dietetic treatment* is important. Raw, clean milk should be substituted for sterilized milk; where this is not possible pasteurized milk should be used. Oatmeal water, or dextrinized gruels may be used as a diluent for the milk; any time after the sixth month orange juice may be given to the normal infant, beginning with the juice of half an orange once in twenty-four hours. The use of cream mixtures, which has been so widely recommended, is attended with some danger; there can be no doubt, however, but that, in infants having a normal digestive capacity for fats, cream, provided it is clean and wholesome, may be added to advantage in small quantities to the milk formula. An infant under six months of age should, however, not exceed 3 per cent. of fat and in older infants the fat percentage should not go above 4 per cent. The thick malt extracts, such as maltine, maltzyme and Trommer's malt, are much more valuable in the treatment of constipation in infancy than cream, and they are not attended with any danger. They may be given in the nursing bottle; for infants three months of age one and a half teaspoonfuls, and at one year of age four teaspoonfuls in the twenty-four hours. With this treatment it is commonly possible to control constipation during the first year of life, but it should ever be kept in mind that if constipation at this age cannot be relieved by simple means it is better to temporize, awaiting the time when the infant is able to take other foods for a permanent cure

of the condition. Many vigorous, healthy infants have been made ill by strong medicines or by feeding cream in too large quantities or by using other foods beyond the digestive capacity of the infant.

Treatment During the Second Year.—*Dietetic.*—All the measures above noted may apply in the treatment of constipation during the second year. Orange juice may be used in larger quantities, cream may be given with less danger, and cooked fruits, such as apple sauce made from ripe apples, and prune juice, may be allowed. Whole wheat bread, well buttered, may be added to the diet and in the latter part of the year fresh, well-cooked green vegetables may be given.

In older children raw and stewed fruits suited to the age and digestive capacity of the child should be prescribed. Bran biscuits, whole wheat flour, unstrained oatmeal with butter and cream and a liberal supply of fresh vegetables should form part of the diet, and the child should be induced to take large quantities of water between meals. Abdominal massage is of great value, especially in older children; it should be of such a character that it will give tone to the abdominal muscles, stimulate peristalsis and facilitate the emptying of the colon and sigmoid flexure. An active outdoor life or gymnastic exercises under a satisfactory tutor should be insisted upon. In obstinate cases Kerley recommends the introduction of four to six ounces of warm olive oil at bedtime; the oil is to be introduced high up into the sigmoid flexure and is to be retained during the night; if necessary a napkin can be used to prevent the child from soiling the bed.

*Medical Treatment.*—Olive oil or codliver oil, combined with one of the thick malt extracts, may be used to great advantage, especially after the first year of life. Calomel and sodium phosphate may occasionally be necessary for the relief of acute conditions which may arise. The sodium phosphate is especially valuable in children over two years of age with a gouty or bilious diathesis, suffering from hereditary constipation. In these cases a saturated solution of phosphate of soda may be given in milk. Sulphate of magnesia and Rochelle salts, like castor oil and rhubarb, are of value only in beginning the treatment of aggravated cases or for relieving acute intestinal symptoms which may develop during the treatment. Cascara is the cathartic of greatest value in the treatment of constipation in older children; the aromatic cascaras on the market are, for the most part, reliable; they should always be given at bedtime and an effort should be made, as time goes on and other treatment is instituted, to gradually reduce the dose. In carrying out this treatment the child should be made to use the chamber at a regular time every morning, and if at the expiration of fifteen minutes a satisfactory evacuation has not resulted a gluten suppository or a small salt water injection should be given. In this way the habit of going to stool at a regular hour will be established and this will do much to complete the cure of the constipation. The habit of regular evacuations at regular hours should be begun in early infancy; it may sometimes be accomplished when the child is six months of age, but from the end of the first year it should be an important part of the routine

treatment and should be insisted upon long after the constipation has been cured. In connection with this method of training and the use of cascara, nux vomica or strychnin may be employed; 4 drops of nux vomica or 1/100 grain of sulphate of strychnin, given three times a day before meals, is a suitable dose for a child six years of age. These drugs, if dissolved in equal parts of sherry wine or essence of pepsin, are not unpalatable.

℞ Strychnin sulph. ..... ¼ gr.
Sherry wine ..... 2 ozs.
Ess. pepsin ..... 2 ozs.
Sig. Teaspoonful before eating for child six years of age.

This prescription may be continued for many weeks, and in the meantime the cascara, which is being given in very accurate and just sufficient doses to produce a normal evacuation, is gradually diminished.

## CONGENITAL DILATATION OF THE COLON

**Etiology.**—Congenital dilatation of the colon is a rare disease which may manifest itself in two rather distinct clinical types.

First, the condition described by Hirschprung in 1880. It has its origin in a congenital malformation of the colon which manifests itself by clinical phenomena directly after birth. The colon is elongated, convoluted and much dilated and its walls are commonly hypertrophied. This condition leads to the accumulation and retention of the meconium and other fecal matter and to a subsequent gaseous fermentation, which results in distending and further dilating the colon. From time to time during the progress of this disease bacteria may cause putrefactive processes which may result in a general systemic intoxication.

The symptom group in these cases is very characteristic. The infant at birth may not appear abnormal, since its intestinal canal is free from bacteria and fermentative processes, but as soon as it commences to take food abdominal distention appears and the attendants note the fact that the bowels have not been evacuated. Rectal injections may cause the discharge of gas and small quantities of fecal matter. The escape of gas flattens the intestine, but the dilatation of the abdomen very shortly returns as a result of the gaseous fermentation going on in the colon. The infant fails to thrive and, as a rule, continues to grow weaker, since in these cases it is practically impossible to keep the colon evacuated and control the fermentative processes. As time goes on, if the infant lives, the distention of the colon becomes so great that it may be outlined against the enormously distended abdomen, and the fecal masses, removed by cathartics and enemata, are covered with mucus and have a putrid odor. Sooner or later—it may be weeks and it may be many months—the infant gradually succumbs from inanition or intestinal intoxication. The latter may produce stupor, coma and convulsions.

The second group of cases also has its origin in a congenital malformation of the colon, which is apparently a great exaggeration of the physiological condition found in infancy, namely, the long-convoluted, freely movable sigmoid flexure and descending colon. The fecal material accumulates in this reservoir and undergoes a gaseous fermentation, and later putrefactive processes may cause intestinal toxemia and start catarrhal processes in the mucous membrane of the cecum, which may end in ulceration and abscess. The differentiation of the two types of congenital dilatation of the colon lies largely in their symptomatology and the time in the life of the infant when the first symptoms are made manifest.

**Symptomatology.**—An infant, that has not perhaps thrived well from birth, at the end of the third or fourth month of life develops a very marked constipation associated with abdominal distention. As time goes on the constipation becomes more aggravated and the colonic distention becomes greater, so that the syndrome produced gradually comes to resemble that of the first group of cases which occurs directly after birth. This second group of cases, however, occurring in older infants, is not so severe in its onset and not quite so serious in its nature. The enormous dilatation of the colon, which in time results, is not altogether congenital, but is for the most part brought about by fermentative processes in the sigmoid flexure. Gradually the colon becomes so enormously dilated that it distends the abdomen to such an extent as to interfere with the action of the diaphragm, even producing dyspnea and cyanosis. As the disease progresses it becomes a more and more difficult matter to unload the colon, and the fecal matter discharged contains more mucus and is more putrid. The intestinal toxemia and inanition present in these cases materially assist in producing the emaciation, the progressive anemia, asthenia and the severe nervous symptoms, such as coma and convulsions, which may mark the downward progress of the disease. A most satisfactory demonstration of the enlarged colon can be made by X-ray pictures, after giving large doses of subnitrate of bismuth; by this method the size and position of the colon can be made out.

**Prognosis.**—The prognosis is bad, only about 10 per cent. of the cases reported have recovered. The disease may be prolonged for a number of years; the majority of cases, however, die before the end of the second year, but some of the milder ones assume a chronic form and may live for eight or ten years. The prognosis in the chronic cases is slightly more favorable.

**Treatment.**—The medical treatment is very unsatisfactory. The prime object is to sustain and nourish the child, if this be possible, by giving it the foods most suitable to its age and condition. In the acute cases occurring just after birth, or within a few months thereafter, breast milk is the only food to be considered. If the infant live, however, longer than one year the dietetic treatment is to be the same as that outlined under Chronic Indigestion. The next indication is to relieve the gaseous distention of the colon by the introduction of a rectal tube and to keep the bowels open and the colon as well washed out as possible by mild laxative medica-

tion and by enemata. In early infancy the most appropriate laxative is the milk of magnesia. In the milder and more chronic cases that live to be over two years of age the cascara preparations are more appropriate. The daily use of salt water enemata, for the purpose of unloading and cleansing the colon, is necessary throughout the course of the disease. In addition to these measures the chronic cases may be benefited by massage and electricity. The massage should begin at the cecum and end at the sigmoid flexure, its object being to unload the colon and to give tone to its muscular coats. Faradization of the large intestine has also been recommended; in applying electricity both electrodes may follow the course of the large intestine as outlined through the abdominal wall, or one may be inserted into the rectum and the other be applied to the abdomen over the course of the large intestine. Tincture of nux vomica or strychnin may be used as a tonic in association with the cascara preparations as directed under the treatment of chronic constipation. The permanent cure of these cases, however, can be hoped for only by operative measures; they may be greatly benefited by the establishment of an artificial anus above the point of greatest colonic distention. This surgical procedure, by largely putting out of function the diseased portion of the colon, enables it to partially recover its tone, and the subsequent final cure of these cases is to be hoped for in the closure of the artificial anus and turning the fecal stream again into the colon, which rest, massage and electricity have placed in a more normal condition. Resection of the diseased portion of the colon itself may be made; this operation, however, has not been tried often enough to determine its ultimate value.

## CHAPTER XXIV

### INTESTINAL PARASITES

Within the last fifty years in this country, worms have, both to the lay and the medical mind, been losing their importance as pathological factors. This is largely due to two reasons. First, with an advancing civilization there has come a higher average of personal and household cleanliness and more sanitary methods of disposing of fecal matter; these improvements in the hygienic surroundings of children have actually diminished the percentage of cases. Second, with an advancing knowledge of diseases of children physicians have learned that the vast majority of symptom groups, which were formerly thought to be due to worms, can now be associated with other definite pathological conditions, and that, with the exception of the hook-worm, these intestinal parasites rarely cause serious or pronounced constitutional symptoms.

## INTESTINAL CESTODES

(*Teniæ, Tapeworms*)

**Varieties.**—The following varieties of tapeworm are found frequently enough in children to deserve study: the tenia saginata, the tenia solium, the tenia elliptica, the hymenolepis nana, and the bothriocephalus latus.

Tenia Saginata.—The tenia saginata, or beef tapeworm, is the form most commonly seen in this country. This worm may be over twenty feet in length, beginning with a small square head, 2 to 3 mm. thick, at the corners of which are suckers containing circles of pigment. With these suckers, which are very powerful, the worm fastens itself to the mucous membrane of the intestine. Behind the head is a still thinner neck, which gradually widens out, presenting a tape-like appearance. The body of this tapeworm is divided into segments, which when sexually mature are approximately 18 mm. long and 7 mm. broad. Each matured segment is filled with a uterus, having a central canal with branches like a tree extending in every direction. These uteri when filled with eggs are very easily discerned, but in the mature segments which have been broken off from the worm and been discharged the eggs are not very plentiful. The eggs are oval or round, have a brownish-yellow color, but no very distinctive individual characteristics. Infection occurs from eating beef containing this parasite. The life cycle of this tapeworm, as of all others, passes through three stages—the egg, the embryo and the worm. The eggs are passed with the segments from the intestinal canal of the human being and contaminate the pastures or other food material of cattle. The cattle taking these eggs into their intestinal canals, they are there developed into the embryo. This embryo, which contains the fully developed head of the tapeworm, escapes through the intestinal wall and lodges in the muscles and other tissues of the animal and there becomes encysted, producing a cysticercus. Cattle thus infected and their meat also are said to be "measly." This "measly" beef containing the embryos, if taken in a raw or imperfectly cooked state, passes into the intestinal canal and there the embryo may fasten itself by its suckers to the walls and draw therefrom its nourishment as it gradually grows into the fully developed tapeworm.

Fig. 32.—Tenia Saginata. (Strumbell.)

Tenia Solium.—Tenia solium, or pork tapeworm, has a very small head with four suckers and a rostellum surrounded by a double row of hooks, 20 to 30 in number. With these hooks as well as with its suckers the pork tapeworm attaches itself to the mucous membrane much more firmly than does the beef tapeworm. Behind the head is a very thin neck passing into a tape-shaped body that is divided into segments, which when mature

are 10 mm. long and 6 mm. broad. The fully developed worm may be 20 feet in length. The uterus, which fills the matured segments, differs from that of the beef tapeworm in that its central canal is heavier than its branches heavier and more irregular in form. The eggs are globular with a

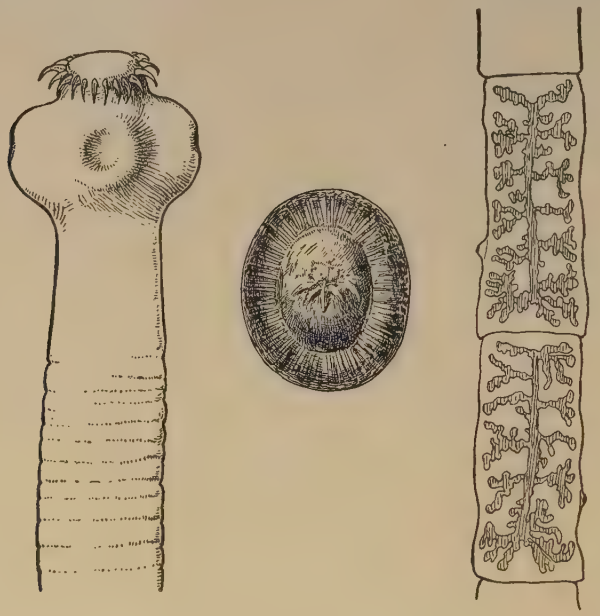


Fig. 33.—Scolex, Egg and Ripe Segments of Tenia Solium. (Wood.)

diameter .03 mm. The life cycle of this tapeworm is similar to that of the beef tapeworm except that its embryo stage is passed in the hog. Infection therefore occurs from eating raw pork.

Hymenolepis Nana.—Hymenolepis nana, or the dwarf tapeworm, is, according to Schloss, a common intestinal parasite in children. He found it in 14 out of 230 dispensary patients. Its average length is 14 to 16 mm.; its distal half is broad, its proximal half narrow. The segments are 3 to 6 times as broad as they are long; the head is globular, carries four suckers and a rostellum with twenty or thirty bifid hooklets. Its eggs are slightly oval and have two widely spaced membranes. From the inner membrane filaments spring which ramify in the space between the membranes. The habitat of this tapeworm is the upper two-thirds of the ileum. Six of the fourteen cases presented no symptoms; in the remaining eight the symptoms were similar to those produced by other tapeworms.

Bothriocephalus Latus.—Bothriocephalus latus, or fish tapeworm, has an oval-shaped head with two elongated depressions serving the purpose of suckers, which attach it to the intestinal canal. It is the largest of all the tapeworms, reaching a length of 30 or 40 feet. The segments or links are only 5 mm. long and about 14 mm. wide, making a characteristic differentiation between this and other tapeworms. The slight length and

great breadth of these segments present to the naked eye a characteristic picture. The uterus is simpler in form, having only 5 or 6 branches. The eggs are oval, yellowish-brown in color, are .07 mm. long and .045 mm. broad, and they have at their top a peculiar cap or lid which has the appearance of closing the egg cavity. The life cycle of this worm is the same as other tapeworms except that its larval or embryo stage is passed in the body of certain fishes; the pike, the perch, the trout, and the salmon are its most common hosts. Infection occurs from eating raw fish thus infected. This worm is most common, therefore, among the fish-eating population along the lakes and seashore.

Tenia Elliptica.—Tenia elliptica, or the dog or cat tapeworm, has upon its head four suckers and a rostellum with fifty or sixty hooks which enable it to attach itself most firmly to the mucous membrane of the intestinal canal. Its links are 8 mm. long and 2 mm. broad. These long and narrow links differentiate it from other tapeworms. It passes its larval stage in the lice of the house-dog or cat, and these insects are swallowed by these animals and can be conveyed to children who play with them. This worm is not of common occurrence, but the great majority of the cases occur in children.

**Symptomatology.**—The habitat of the full-grown tapeworm is in the intestine, and the vague symptom group which it causes comes largely from the irritation which it there produces. Indigestion, nausea, headache, nervous irritability, sleeplessness, and in rare instances more severe nervous symptoms may result. This symptom group, however, has nothing whatever characteristic in it, since in the majority of instances it is almost or quite absent, the child appearing in normal health. In a very small per cent. of the cases of Bothriocephalus latus there occurs a very severe form of anemia. Eosinophilia and a mild simple anemia occur in nearly all children who have had tapeworm for any length of time.

**Diagnosis.**—Diagnosis in nearly every instance is made by seeing the segments of the worms in the stools. The parents or nurses can scarcely overlook for any length of time the tapeworm segments; as they mature they are broken off and escape by the rectum. The cases, therefore, come to the physician with a diagnosis made. If, however, there be any doubt an active cathartic may be given to carry away segments of the worm. Failing in this the fecal matter may be examined microscopically for the eggs.

**Differential Diagnosis.**—The differential diagnosis of the varieties of tapeworm can, as a rule, be made by carefully studying the ripe segments, and may be of considerable importance, especially between the tenia solium and the tenia saginata, the two most common varieties. If the tenia solium be present the treatment is more urgent and should be more energetic, since it fastens itself more tightly to the intestinal canal by hooklets, and there is also greater danger in this form from cysticerci or the encysted embryos, which may result from the eggs of tenia solium, finding their way into the stomach. In such instances the child becomes the intermediary host, and the embryo, formed in its intestinal canal from the egg, passes through the

intestinal wall and finds a lodgment in the muscles, brain, or other organs, where it may produce disease even after the intestinal canal of the child has been entirely cleared of tapeworms.

**Treatment.**—The remedies used for the treatment of tapeworm are more or less toxic and more or less irritating to the gastrointestinal canal. They should therefore be used with caution, and in every case the question must be decided whether the child is in a proper physical condition to undergo the treatment. In all cases where the child is acutely ill from other diseases, especially from gastrointestinal troubles, and in all cases where it is weak and malnourished from some other chronic disease, it is better to postpone the treatment until it can at least be put in a fair condition of health. During this time the child should, at intervals of four or five days, be given doses of castor oil for the purpose of breaking off sections of the worm and discharging them. In this way the intestinal irritation may be modified and the danger of cysticercus minimized.

The Cure.—After a day of preparatory treatment, during which the child has little to eat and is given at bedtime a saline laxative, the medicines for stupefying and expelling the worm are given early the next morning on an empty stomach. In this country the oleoresin of aspidium (filix mas) has been very generally and very successfully used. It is perhaps the most satisfactory of all remedies. It should be given in doses of from 7 to 30 minims, depending upon the age of the child. In infancy it is advisable to begin with from 5- to 7-minim doses; if this treatment fails, at its next administration the dose may be increased; for a child three years of age 10 to 15 minims may be given; for a child six years of age 30 minims. Two doses of the size above noted should be administered, one when the child awakens in the morning and one an hour later. The child should then be kept as quiet as possible without food. Four or five hours after the last dose of male fern a saline laxative should be administered. This is to be preferred to castor oil, as it is less likely to provoke vomiting and as there is a suspicion that oil increases the toxic action of filix mas. One or two hours after the laxative small quantities of food in the form of beef broth may be commenced. This treatment commonly results in the expulsion of the entire worm. A careful examination should be made by the nurse of the fecal matter expelled and every particle of the worm carefully saved for the physician's examination. The object of this examination is to find the head of the tapeworm; if this be found a cure may be assumed, since, as a rule, only one worm is present at a time. If the head is not found one is always in doubt, and two weeks later the same treatment may be repeated. If the head is not then found it is advisable to wait until time determines whether the child has been cured or not. If after a number of weeks the segments again appear in the stool, the same treatment is repeated, giving one-third larger dose of the male fern. It is not commonly necessary to repeat this cure again, since this treatment in the great majority of instances is successful. Following the giving of the male fern and the cathartic, the child should for a few days be carefully dieted to avoid gastro-

intestinal irritation. The best form for administering oleoresin of aspidium will depend upon the age of the child. If the child is old enough it may be given in capsules, if not, in an emulsion of equal parts of gum tragacanth and simple elixir. A favorable vehicle is thus offered for the administration of a drug which not only is very distasteful to the child, but may produce nausea and vomiting and thus necessarily postpone the cure.

Prophylactic Treatment.—As rare beef, and beef juice made from raw beef, are such common articles of diet with young children, it is wise to explain to parents that there is a slight danger of contracting tapeworm from eating these foods. This danger can be removed by stopping these foods and permitting children to have only beef, pork and fish that have been well cooked, or it may be minimized by carefully scrutinizing all meat for cysticerci, before preparing raw foods for children. This latter method of prophylaxis with reference to rare beef and beef juice is to be advised because there is only the slightest possible danger, with this precaution, that the child may contract tapeworm, and the disease itself is not of sufficient seriousness to warrant the elimination from the child's diet of two of the most important foods during this period of growth and development. Nevertheless, as a fundamental principle of prophylaxis one should advise, with these exceptions, that all meat foods should be well cooked.

To prevent the spread of this disease by those infected, it is necessary that their fecal discharges be carefully disposed of. In the city this may be done by giving great care to the personal cleanliness of the child, burning the segments as they appear in the stools, scalding out all vessels used for the reception of fecal matter, and flushing the feces into the sewer as soon as possible. In the country, where sanitary plumbing is not used, it is best to burn all fecal discharges.

## ASCARIS LUMBRICOIDES

**Characteristics.**—This is the common round-worm, has a pale red color, is cylindrical in shape and has pointed extremities, resembling the ordinary fishing worm. The male is about 20 cm. long and 4 mm. thick and its tail is curled up over its abdomen. The female is larger, being about 30 cm. long and 5 mm. thick. The female produces millions of eggs which are disseminated in great profusion through the fecal contents of the intestinal canal. The eggs measure from .05 to .06 mm. in length and are both round and oval in shape. The embryos develop only from the round eggs; the oval form is unfruitful. The outer layer of these eggs has a rough nodular surface and is yellowish-brown in color. The life cycle of the round-worm does not require an intermediate host. The eggs propagate rapidly in moist earth, so that the surroundings of the patient may be readily contaminated from the intestinal discharges. This leads to the spread of this disease among the uncleanly, among whom this condition is most frequently found. More than one worm is commonly present and they may exist in great numbers, so great, in fact, that they may entwine

themselves in masses of sufficient size to produce intestinal obstruction. They may migrate to all portions of the gastrointestinal canal, appearing in the stomach and producing gastric irritation and vomiting; occasionally finding their way into the larynx, trachea and bronchi, producing there serious symptoms of obstruction. They may enter the bile duct, causing jaundice and even abscess of the liver, and by penetrating into the appendix may produce appendicitis.

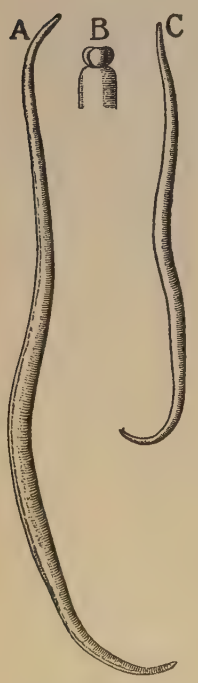



Fig. 34.—Ascaris Lumbricoides, Female, Head and Male.

**Symptomatology.**—In the great majority of instances constitutional symptoms are absent. But there may be more or less intestinal irritation, which in young and delicate children is marked by digestive disturbances and mild nervous symptoms, largely reflex in their nature. The appetite may be lost or perverted, slight nausea and diarrhea may occur, and the child may be restless, irritable, sleepless and complain of headache. Picking at the nose and rectal irritation are common, but not at all characteristic, symptoms. In rare instances more severe nervous symptoms, such as convulsions, may occur. The writer observed one such instance in an apparently normal child, eight years of age, who lay in convulsions for eight or ten hours, and was immediately relieved and at once convalescent on the passage of a large ball of tightly matted round-worms. The convulsions in such cases may in part be the result of the intestinal obstruction and resulting intoxication which the ball of worms produces, or they may be caused, as the French writers believe, from the poisons which the round-worm excretes into the intestinal canal. Eosinophilia may be associated with the presence of these worms.

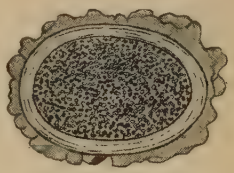


Fig. 35.—Egg of Ascaris Lumbricoides. (Wood.)

**Diagnosis.**—The above symptom group varies so greatly in different cases and marked symptoms are so commonly absent, that the diagnosis of round-worms is ordinarily made by seeing the worms in the intestinal discharges. When this disease exists, a cathartic, such as castor oil, will almost certainly reveal the presence of the worms in the stools, but a much more accurate method of diagnosis is to examine the feces under the microscope. The eggs will always be found in abundance. This latter method of examination is so reliable that it is advisable, following the treatment for this condition, to make a second microscopical examination of the feces to determine whether the eggs have entirely disappeared.

**Treatment.**—Santonin is a specific for this condition. It is advisable to either combine it or follow it with calomel, and some hours later by a

saline laxative or by castor oil. Two doses of the santonin and calomel should be given, the first dose on an empty stomach before breakfast, and the second at bed-time, to be followed the next morning by the castor oil. The dose of santonin is one-half grain for a child one year of age and one grain for a five-year-old child. Larger doses may produce poisoning. This treatment may be repeated after an interval of one week, and in the great majority of instances a cure is effected. The following prescription offers a suitable form for the administration of this drug:

℞ Santonin ..... grs. i
Calomel ..... grs. i
Sacchari albi..... grs. vi
M. Div. in chart No. 2.
Sig. One before breakfast and one after supper for a child one year of age.

All of the ingredients of this prescription may be doubled for a child five years of age.

*Trichuris Trichiura.*—Trichuris trichiura, or the whip-worm, is a common intestinal parasite. It is a whip-shaped round worm, about two inches in length, which has its habitat in the large intestine. It is of little clinical or pathological importance, since it rarely produces either constitutional or local symptoms.

## OXYURIS VERMICULARIS

**Characteristics.**—Oxyuris vermicularis is the ordinary thread-worm. It is not, like the round-worm, so largely confined to the uncleanly, but may occur among all classes of society. It is very small and looks not unlike a piece of white thread; is spindle-shaped and white in color. The male is between 3 and 4 mm. long, and the tail is curved toward the abdominal surface. The female is much larger, 9 or 10 mm. in length and two or three times as thick as the male, but its tail is not curved forward. The female produces thousands of eggs, oval in shape and about .05 mm. long. The eggs, however, of this species are not so important from a diagnostic standpoint as they are in the round-worm. The thread-worm requires no intermediate host. The child may reinfect itself or spread the disease among other children after contaminating its fingers by scratching the anus. The female lives in the large intestine, and the embryos make their way into the small intestine where the males predominate, but as they mature both males and females migrate to the large intestine. As the eggs and worms are discharged with the feces the surroundings of the child are contaminated. This offers a favorable opportunity not only for the reinfection of the child, but for the spread of the disease to other children.

**Symptomatology.**—The irritation which these worms produce in the colon may result in mucous discharges. Pruritus ani is the most common and the most troublesome symptom. The intense itching of the rectum, which is much worse at night, is due to the fact that the worms migrate at

this time to the outer rectal folds and they may there be seen by pressing apart the folds of the anus. This itching of the rectum causes the child to be sleepless and irritable, and results in scratching and tearing the part with the fingers; this is usually done during sleep. The traumas resulting from scratching very commonly produce eczema and scars about the anus. Thread-worms may be the reflex exciting cause of nocturnal incontinence of urine, pseudo-masturbation, and night terrors. Loss of weight, anemia and headache may occur. The most serious localized disturbance which can be produced by these worms in the large intestine is appendicitis, but this is a rare occurrence.

Fig. 36.—Oxyioris Vermicularis, Female, Immature Female and Male.

**Diagnosis.**—The symptom group above outlined at least suggests the presence of the thread-worm, and the diagnosis is confirmed by making an examination of the anus. For reasons above given this is more successful if the inspection is made some hours after the child has gone to bed. If the worms are not found in the rectal folds an enema should be given, and in the resulting discharge the worms can be seen. A dose of castor oil will also answer the purpose of bringing these parasites to light.

**Treatment.**—The most effective remedy is flushing the colon with normal salt solution. High injections that completely irrigate and wash the mucus out of this organ are necessary to successful treatment. These irrigations should be followed by 6 to 10 ounces of infusion of quassia, injected high into the colon; this should be repeated every day for four or five days, and after a period of four or five days' rest the irrigation may again be resorted to. It may be necessary in obstinate cases to repeat this treatment as many as four or five times, but the great majority of cases are cured after the second or third course of injections. Each time before beginning the irrigation it is advisable to give a preliminary dose of calomel and santonin, of each one-half grain, followed by castor oil or some other cathartic. This treatment serves the purpose of driving these parasites out of the small intestine where they can be reached by the colon irrigation.

In rare instances one comes in contact with a case of thread-worms that fails to respond to treatment. Some of these cases may be due to a lack of prophylactic treatment in connection with the cure above given. The ova are so commonly found under the finger nails as a result of scratching, that in every case the greatest cleanliness should be observed. The child's hands and finger nails should be kept clean with soap and water, and in obstinate cases the child should sleep with closed pajamas so as

to keep the fingers from coming in contact with the rectum during sleep. Fecal discharges should be disposed of with the same care and in the same manner as just described under the treatment for Ascaris Lumbricoides.

## CHAPTER XXV

### INTESTINAL INTUSSUSCEPTION

Intestinal intussusception is the invagination of one part of the intestine by another; the upper portion of the gut commonly slips into the lower, but the reverse of this may take place, the lower portion slipping into the upper. This produces a cylindrical tumor composed of three parallel layers of intestine. This tumor may itself again be invaginated or swallowed up by the neighboring intestine; this double invagination is much more likely to occur where the small intestine alone is involved in the process. In intestinal invagination the mesentery is also swallowed up and drags at the head of the invaginated gut; this greatly increases the traumatism and causes inflammatory processes in the affected part; it also aids materially in producing the more or less complete obstruction of the bowels, since the dragging of the invaginated mesentery pulls the invaginated gut out of line with the intestinal canal.

Intussusception may occur throughout the intestinal canal, but in the vast majority of instances it involves some portion of the ileum or colon or both, and the different varieties depend upon the portion of the intestine involved. The ileocecal type is the most common; it includes 70 or 80 per cent. of all cases; in this form the colon swallows up the cecum and ileum; the ileocecal valve preceding the mass may reach the rectum. The ileocolic type embraces from 10 to 15 per cent. of the cases; in this form the ileum slips through the ileocecal valve, the valve remaining in position. The colic type embraces 2 or 3 per cent. of the cases; in this condition the colon slips into itself, and passes downward toward the rectum. The ileac type embraces from 5 to 8 per cent.; in this condition the small intestine is telescoped into itself and this commonly occurs in the ileum.

**Etiology.**—Age is an important predisposing factor, as intussusception occurs in children very much more commonly than it does in adults. It is observed most frequently in the second half of the first year and diminishes thereafter, but is not uncommon up to the sixth or seventh year. It is by far the most common form of intestinal obstruction observed in childhood. It occurs more frequently in weak, malnourished children, and especially in those that have suffered from previous intestinal disease; in these children the musculature of the intestine is weak, irritable and subject to abnormal, irregular, peristaltic contractions, which are the great exciting causes of this disease. These abnormalities in peristalsis may be excited by constipation, intestinal disorders, improper food, injury to the

abdomen and irritations and inflammations of Meckel's diverticulum and the appendix.

**Pathology.**—As previously noted, the swallowing of both mesentery and intestine increases the engorgement, aggravates the strangulation and produces complete intestinal obstruction which results in necrosis and gangrene of the invaginated gut. Snow, of Buffalo, reported a case in an infant seven months of age, who for sixteen days had had symptoms of intussusception; six inches of gangrenous intestine which protruded from the rectum was removed and the infant recovered. In the chronic cases the obstruction is not complete, but the invaginated intestine is held firmly in position by inflammatory tissues.

**Symptomatology.**—General Symptoms.—This disease begins suddenly, not uncommonly during sleep, with severe intestinal pain which recurs in paroxysms. The pain is associated with vomiting, usually severe and persistent in character, and, after a few hours, with the passage from the bowels of bloody mucus and very little or no fecal matter. This symptom group is practically characteristic of the disease, and of great importance is the fact that these symptoms are associated in the beginning with little or no fever.

The *pain* is similar to that which occurs in severe intestinal colic, the infant cries loudly, draws its legs upon its abdomen and squirms with pain; after a time the paroxysm disappears, only to return with the same severity after a longer or shorter interval. The attacks of pain are supposed to be associated with violent peristalsis and perhaps to mark the various steps in the progress of the invagination. After a few hours they become less severe and from this time on the intestinal colic is no longer a prominent symptom of the disease.

*Vomiting* occurs early and very frequently continues throughout the course of the disease. The intervals between the paroxysms may be measured by minutes or by hours. The vomited matter consists first of food, then of mucus, perhaps stained with blood, then bile, and in some instances fecal matter; this is more marked in older children and is associated with complete intestinal obstruction. In a small percentage of the cases vomiting is not so prominent a symptom; it may come on late and recur only once or twice in twenty-four hours.

*Intestinal Discharges.*—The bloody mucus which is discharged in 80 per cent. of the cases, and commonly within the first twenty-four hours, is our most valuable symptom in the diagnosis of this disease. Gibson regards it when taken in connection with the other symptoms as pathognomonic. The blood in rare instances is discharged in large quantities; it usually, however, occurs in such small quantities as only to slightly tinge the mucous discharges. These discharges of bloody mucus may be associated with tenesmus and relaxation of the rectal sphincter when the intussusception approaches the rectum. With the onset of this condition the first intestinal discharge may be normal, followed by slightly loose movements, which within the first twenty-four hours consist almost entirely of bloody

mucus, with little or no fecal matter. With the increase in the obstruction, which commonly becomes complete, the discharge of gas and fecal matter ceases. In some instances, however, small quantities of fecal matter and gas may escape through the invaginated intestine for several days. In the rare cases of chronic intestinal obstruction occurring in older children, gas and fecal matter continue to be discharged throughout the disease. When gangrene occurs the discharges may contain shreds of putrid, foul-smelling tissue.

*Fever.*—In the beginning intestinal intussusception is an afebrile condition. After the second or third day there may be a slight rise in temperature and if the child lives for four or five days the temperature rises as a result either of intestinal infection or of peritonitis.

Physical Examination.—A tumor, which can be found in from 40 to 50 per cent. of these cases, should be carefully searched for; abdominal palpation, however, should be made when the child is free from pain and the abdomen completely relaxed; anesthesia may be necessary in some cases; the tumor, which is an elongated tender mass, is commonly found in the transverse or descending colon. The presence of localized tenderness may assist in the localization of the tumor. The examination per rectum with the finger, in 30 to 40 per cent. of the cases, will reveal the invaginated intestine which has pushed down almost or quite into the rectum and the examining finger on withdrawal may be covered with bloody mucus. This examination should be made in every instance, since the tumor after all is the only pathognomonic sign of this disease. The general appearance of the child is of value in the diagnosis; in severe cases there is marked prostration, and the infant has a pale, anxious expression, which during an attack of pain may be associated with cyanosis and with a rapid feeble pulse.

Course.—The course of this disease may be very acute, the child dying within the first twenty-four hours; these rapidly fatal cases, however, are very rare. In most instances it lasts from a week to ten days; it reaches the crisis more rapidly in infants than it does in older children. When the case becomes chronic it may be prolonged indefinitely.

**Prognosis.**—This depends largely upon an early diagnosis and early surgical interference; it is much more favorable in infants than in older children; if the diagnosis is made in the first twenty-four hours more than 60 per cent. recover. The average mortality according to Gibson is 53 per cent.

**Diagnosis.**—This is made by the sudden onset of a severe intestinal colic, associated with vomiting and with the passage of bloody mucus, but with the discharge of little or no fecal matter. This symptom group occurring as an *afebrile* condition is sufficient to warrant an exploratory laparotomy, even if the physical examination fails to reveal a tumor. The demonstration of the tumor, however, either by abdominal palpation or rectal examination, makes the diagnosis certain. This disease should not be confounded with enterocolitis, which is from the onset a distinctly

febrile disease presenting an entirely different symptom group. It resembles this disease only in the passage per rectum of bloody mucus. Acute appendicitis may, by its vomiting, pain and obstinate constipation, suggest intussusception. In this condition, however, the bloody stools and tumor are absent, and the disease is distinctly febrile from its onset. The further points in the differential diagnosis of this condition may be made out by a study of the symptoms of appendicitis.

The diagnosis of intussusception from other forms of intestinal obstruction is, as a rule, not difficult, and in this differentiation practically only two conditions have to be considered, viz., obstruction from fecal impaction and from inflammatory bands caused by appendicitis or peritonitis. The diagnosis of fecal impaction may be made out by rectal examination and by physical examination of the abdomen, as well as by the history of the case and the results of the preliminary salt-water enema. Obstruction from inflammatory bands may be determined by the complete occlusion (no feces or gas), the severity of the vomiting, the absence of bloody mucous discharges, and the presence of appendicitis or peritonitis.

**Treatment.**—No time should be lost in obtaining surgical relief, as there is little to be hoped for from any other line of treatment, and much to be lost by postponing surgical treatment in the hope that less radical measures will relieve the condition. All writers agree, however, that no harm can come from the introduction into the colon of large quantities (1 quart) of warm normal salt solution. In giving this enema the child's hips should be elevated and a rectal tube inserted into the sigmoid flexure, and through this tube, from the bag which is elevated not more than two feet above the patient, the water is allowed to flow. In some instances relief has followed. The forcible introduction of water, oil, air or gas, for the purpose of reducing the intussusception, is accompanied by more or less danger; rupture of the intestine has occurred during these manipulations.

By an exploratory laparotomy the surgeon may determine the character of the operation to be made. It may be found that a simple reduction of the intussusception is all that is necessary, or it may be necessary to shorten the mesentery at the site of the intussusception to prevent its recurrence, and in other instances a resection of the gut may be obligatory. All forms of intestinal obstruction, except that produced by fecal impaction, are essentially surgical.

## CHAPTER XXVI

### APPENDICITIS

The term appendicitis, as now used, includes periappendicitis, typhlitis, perityphlitis and localized abscess and peritonitis occurring in the appendicular region.

**Etiology.**—The disease is rare in infancy, but after the second year of life becomes much more frequent and during childhood is not uncommon. Heredity, as Forchheimer has shown, is an important etiological factor. The hereditary predisposition may depend upon inherited anatomical peculiarities of the appendix, such as its length, location and patulency, or upon the character of lymphoid tissue associated with it, or on an hereditary tendency to constipation; at any rate it is a fact that this condition may be a "family" disease running through various generations. Among the exciting causes are constipation, especially fecal impactions in the cecum; colitis involving the cecum and extending to the appendix; intestinal infections which destroy the normal intestinal flora and substitute therefor pathogenic flora, such as the bacillus coli, streptococci and staphylococci; foreign materials such as fecal concretions; seeds and undigested food; intestinal worms; blows upon the abdomen, or unusual muscular exertion lighting up a latent inflammatory condition; and lastly, scarlatina, typhoid fever, tonsillitis and intestinal grippe. These last-named infections may be associated with cases of appendicitis. Of these exciting causes bacteria are undoubtedly of the greatest importance.

**Pathology.**—The appendix in the child is commonly located lower down in the pelvis than it is in the adult. This position of the appendix and cecum is of importance, in that in the physical examination of these parts the enlarged appendix, or tumor masses resulting from appendicitis, must be felt for lower down and often under the anterior spine of the ilium. This position of the appendix, often in the little pelvis, may direct the burrowing abscess deep down into the floor of the pelvis and up on the other side in close association with the rectum. The location of the appendix, however, in the child is even more variable than it is in the adult; it not infrequently is directed upward and lies back of the cecum. The variability in the location of the appendix determines the various locations of the abscess and inflammatory thickenings which occur in this condition.

The forms of appendicitis in children are similar to those occurring in the adult: the catarrhal, the ulcerative and the gangrenous. There is, however, on the part of the child an apparent predisposition to the ulcerative or perforative variety, since these cases occur in relatively larger proportion. In the catarrhal form the mucous membrane of the appendix is congested, inflamed, and its lumen distended with mucus or mucopus. In these cases the contents of the appendix are discharged into the cecum and there produce more or less irritation, aggravating an old colitis, which may have been the original cause of the appendicitis. At any rate colon infection and attacks of subacute colitis occur in these cases, and the persistent factor in many a chronic case of chronic colitis is an existing catarrhal appendicitis. In the ulcerating or perforating form, the mucous membrane of the appendix becomes infiltrated and finally breaks down under the infection and distention. The perforation generally occurs near the end of the appendix, which not uncommonly holds a plug of hard fecal mat-

ter or a quantity of mucus and pus, which by their pressure have facilitated the rupture. In the gangrenous variety a portion of the distal end of the appendix becomes necrotic and sloughs off; this process, which is perhaps due to some disturbance of circulation, allows the contents of the appendix to be discharged through the opening produced by the slough. The fecal and other concretions present in both the ulcerative and gangrenous types may be an effect rather than a cause of the appendicitis. It is probable that most cases of appendicitis would remain catarrhal if there were a free opening through which the contents of the appendix could be discharged into the colon, but with the congestion of the mucous membrane this opening, at all times insufficient, becomes more tightly closed, shutting up within the small lumen of the appendix the mucus and pus formed by the inflammatory process. Various microorganisms, especially the colon bacillus, streptococci and staphylococci, play an important rôle in the etiology and pathology of the more severe cases.

In the ulcerative and gangrenous forms of appendicitis the appendix is commonly walled off by an inflammatory exudation which catches and holds, at least temporarily, the infectious material discharged from the appendix. This localized abscess thus formed may burrow into the pelvis as previously described, may make its way up behind the cecum or may even present itself anteriorly, perforating the skin; it more commonly, however, breaks through the inflammatory wall which surrounds it into the general peritoneal cavity, producing septic peritonitis. General peritonitis may immediately follow rupture of the appendix, when nature has not had time to wall off an inflammatory pocket for the reception of the infectious material; this occurs most commonly in the gangrenous forms. Inflammatory bands, which are thrown out during the acute process and commonly remain for some time after recovery from the attack, may catch and strangle loops of intestine, thus complicating the appendicitis with an acute intestinal obstruction. This is a danger to be watched for during the convalescence of operative cases, and is much more common in childhood than in adult life.

**Symptomatology.**—This disease may follow an indigestion, or be coincident with an intestinal infection. The initial symptoms are severe abdominal pain, colicky in character, recurring at intervals, commonly associated with vomiting, which may or may not be repeated. Constipation, obstinate in character, is the rule; diarrhea may occur; fever is present. This symptom group occurs with such clearness as to at least suggest the possibility of appendicitis, which should lead to a careful physical examination, upon the results of which the diagnosis is ordinarily made.

Physical Examination.—The child commonly assumes the recumbent posture and lies with its legs flexed upon the abdomen. By gentle palpation, localized tenderness may be located at McBurney's point, but, as a rule, it is lower down, on or below the level of the superior iliac spine. The amount of tenderness on pressure is to be estimated largely by watching the child's face during examination; the facial expression is more

reliable than the child's answers in determining the question of localized tenderness. This examination also makes out the presence or absence of localized resistance of the muscles in the right iliac region; this localized muscular tension is of the very greatest value in the diagnosis, when present in association with the symptom group above given it indicates a rather active appendicitis; this is especially true in young children. A tumor or thickening of the tissues around the appendix, or the enlarged appendix itself, may be made out by careful bimanual examination. In making this manipulation the fingers of the left hand are pressed deeply into the back opposite McBurney's point, and the right hand in opposition to it is pressed gently down into the region of the appendix, carefully palpating the entire region, especially low down in the pelvis. In older children the introduction of the finger high up into the rectum may localize on the right an indurated mass.

General Symptoms.—The *pain* and *tenderness,* however, which mark the onset of this disease commonly continue to be important symptoms, becoming rather less severe but more constant in character. The sudden cessation of pain, which is not infrequently associated with a fall in temperature, is an ominous symptom, especially when the pulse rate continues rapid and the child's general condition is not improved. This symptom group means rupture of the appendix with the relief of tension and the discharge of the infected material into the periappendicular region; this accident is so common that the symptom group which marks it must ever be kept in mind.

*Constipation,* when very aggravated, is commonly associated with vomiting, and this symptom group may suggest intestinal obstruction. The vomiting is more marked following rupture of the appendix and subsequent involvement of the peritoneum.

*Fever.*—A rise of temperature occurs early in this disease and its height may mark the severity of the process. This rule is not, however, without exception, as in rare instances cases of gangrenous and perforative appendicitis may for the first few days run an almost afebrile course. Following the rupture of the appendix and periappendicular infection, there is a secondary rise in temperature which marks the progress of the sepsis or peritoneal inflammation.

Blood Examinations.—The leukocyte count is of very great value not only in confirming the diagnosis of appendicitis, but also in helping to determine whether the disease is progressing favorably or unfavorably. In interpreting these blood counts, however, it should be remembered that in very young children the proportion of lymphocytes is much greater than it is in older children. There is in appendicitis, except in the very worst cases where resistance to the infection is almost lost, a marked leukocytosis of 12,000 to 30,000. This increase in the number of leukocytes is commonly in proportion to the activity of the process; in mild cases the leukocyte count may be 12,000; in septic cases during the acute stage it may run to 30,000. A high leukocyte count is commonly an indication

of pus. Daily blood examinations should be made in all cases where the diagnosis is doubtful, or where an operation for any reason is postponed; an increasing leukocyte count is an unfavorable sign and indicates progress in the inflammation; a falling leukocyte count with other symptoms favorable is a good sign, and is an indication that the child will recover from the present attack. A low leukocyte count occurs in fatal cases toward the end of the disease, but the low leukocyte count is here associated with septic temperature, profound prostration, a rapid, irregular and increasing pulse rate, and is an indication that the vital powers of the child are no longer able to call forth an army of leukocytes with which to fight the infection. A differential leukocyte count is also of value in determining the character of the process. When the polynuclear leukocytes are present in a percentage greater than 80, pus is probably present and this probability is greatly strengthened if the general leukocyte count is above 14,000.

Course.—The course of this disease depends largely upon the severity and character of the inflammation affecting the appendix. In the catarrhal form the symptoms previously noted may be mild in their onset and the disease may run its course within a week or ten days, terminating in recovery. In the ulcerative form the onset is commonly much more severe, the pain, fever, vomiting and localized tenderness being very marked and increasing in severity, until, perhaps between the third and fifth day, perforation occurs, producing a cessation of pain, fall in temperature and otherwise modifying the symptom group as previously noted. The subsequent history of these cases depends upon whether a general septicopyemia or a general peritonitis follows the rupture, or whether the infectious material is walled off from the peritoneal cavity by an inflammatory exudate. In the former case the child quickly succumbs unless perchance it be saved by a surgical operation. In the latter the localized abscess may after a number of days begin to show evidences of resolution and a slow convalescence follows, or it may burrow or break into the surrounding tissues and place the child's life in immediate jeopardy. In the gangrenous form the initial symptoms are even more violent than in the ordinary perforative form; necrosis and general infection may occur on the second or third day of the disease.

**Diagnosis.**—This disease may be differentiated from intestinal obstruction by the presence of fever, the less severe pain, the absence of persistent vomiting and by the presence of the local symptoms of appendicitis made out by physical examination, and by the absence of bloody mucus from the intestinal discharges. Lobar pneumonia of the right lower lobe often presents a picture closely resembling appendicitis; in this condition there may be abdominal distention and pain and tenderness in the region of the appendix, but the absence of the other physical signs of appendicitis, with the presence of the physical signs of pneumonia, should be sufficient to prevent this mistake in diagnosis, provided the physician has in mind the fact that such a symptom complex may be produced by pneumonia.

Typhoid fever may be differentiated by the character of the fever, the presence of the Widal reaction and by the absence of the intestinal colic.

Tuberculosis of the lymphatic tissues of the appendix may produce tumor masses in this region, which should not be mistaken for appendicitis. The history and course of the process which produced the appendicular tumor in tuberculosis is very different from the course of acute appendicitis.

**Prognosis.**—The prognosis in private cases which are seen early and which have the benefit of surgical interference at the proper time is good; more than 95 per cent. of these cases recover. The results that are obtained from surgical interference in the suppurative and gangrenous cases in children are much better than they are in adults. The high death rate (14 per cent.) which occurs in hospital cases is due to the fact that they do not always have the benefit of surgical interference at the proper time.

**Treatment.**—The medical treatment demands that they be kept absolutely quiet in bed, with a total abstinence from food for two or three days, but water may be freely given. On the third or fourth day meat broths and small quantities of whiskey, well diluted, may be allowed. Cathartics or high rectal injections for the purpose of moving the bowels are to be avoided during the early acute stage of this disease. A light ice-bag placed for the greater portion of the time over the appendicular region is a valuable remedy; if this produces discomfort hot applications may be substituted, especially in very young children. Opium may be necessary for the relief of pain, but its use in children is not followed by the same good results seen in adults. When the pain of this disease is severe enough to demand opium it is better practice to refer the case to the surgeon. On the fourth or fifth day, with the subsidence of the symptoms, an enema followed by a dose of castor oil may be given, and thereafter the child may be allowed meat broth, beef juice, albumin water, and later milk. It should be kept quietly in bed until convalescence is assured; relapses during this period are especially dangerous. Following an attack the child should be carefully fed within the limits of its digestive capacity, violent exercise should not be allowed for some months, constipation should be carefully avoided. These precautions are necessary to prevent a second attack.

In the milder or catarrhal forms of appendicitis it is the duty of the physician to try to carry the child through the attack and refer it to the surgeon for an interval operation; this is much better practice than operating upon every case as soon as a diagnosis is made. Under medical treatment 95 per cent. of these cases recover from the first attack, and this mortality would not be diminished by operative interference during the attack. The treatment of the appendicular attack in the great majority of cases is purely medical and in the handling of this phase of the treatment the physician has perhaps more experience than the surgeon. During the treatment of any case of appendicitis, however mild, the physician must realize that at any time the case may become a surgical one demanding im-

mediate operative measures. For this reason the danger signals above outlined must be carefully kept in mind. In the more severe types of appendicitis, when the initial symptoms indicate that one is in the presence of a suppurative or gangrenous appendicitis, the child should, in the midst of the attack, be referred to the surgeon; no possible good can come from delaying surgical interference in these cases, and a difference of twenty-four or thirty-six hours may materially diminish the child's chances for recovery. Every well-defined case of appendicitis should sooner or later come into the hands of the surgeon. The only valid reason for postponing indefinitely surgical interference is that there may have been a mistake in diagnosis; in such cases one should await the subsequent history of the child to confirm or deny the diagnosis.

## CHAPTER XXVII

### PERITONITIS AND ASCITES

### PERITONITIS

**Etiology.**—Appendicitis is the most common cause of acute peritonitis, and tuberculosis is the only cause of chronic peritonitis in childhood. Acute peritonitis from other causes, though comparatively infrequent, is met with often enough to deserve careful consideration.

The MICROORGANISMS most commonly associated with acute peritonitis are the bacterium coli, the streptococcus pyogenes and pyocyaneus, the staphylococcus aureus, the pneumococcus, the gonococcus, the diplococcus intestinalis, and with the chronic form of this disease the tubercle bacillus, assisted in its destructive process by streptococci, staphylococci, and other organisms. These various microorganisms are associated with more or less distinct types of this disease. The bacterium coli communis occurs especially in the forms of acute peritonitis which have developed after intestinal perforation. Streptococci and staphylococci occur in the perforative form and in the septic types of this disease, and also in the forms of peritonitis that follow the infective fevers, such as erysipelas, acute tonsillitis, diphtheria, influenza, scarlet fever, and measles. Pneumococcus peritonitis may occur as one of the localizations of a general pneumococcus infection, or it may spread from a pneumococcus inflammation of the lungs and pleura. Gonococcus peritonitis is also almost always secondary to gonococcus vaginitis. The gonococci entering through the uterus and fallopian tubes, or in any manner finding their way into the pelvic tissues, start up there a local inflammation of the pelvic peritoneum, which may or may not become a general peritonitis.

EXCITING CAUSES.—Appendicitis is the most important cause of acute peritonitis and its exciting causes become, therefore, the common exciting causes of peritonitis. Very rarely typhoid fever, tuberculosis, and dysen-

tery may cause perforation and produce peritonitis. A perforating ulcer of the stomach and duodenum is one of the rarest causes. Suppurative processes in the liver and gall-bladder, strangulated hernia, intestinal obstruction, blows upon the abdomen, perforating wounds from gunshot injuries or sharp instruments, and operative measures in the peritoneal cavity may produce peritonitis. The acute infectious diseases previously mentioned, however, are responsible for a majority of the cases that cannot be traced directly to appendicitis or tuberculosis. In the peritonitis which occurs in the newly born infant the infection is either a septic one entering through the umbilicus or it is due to an enterocolitis which furnishes a favorable opportunity for the infection of mesenteric lymph nodes and later of the peritoneum.

**Pathology.**—The pathological process varies largely with the character of the inflammation. In all cases this membrane is more or less congested and covered with a serous or fibrinous exudate. In the fibrinous cases the coils of the intestine and the omentum are frequently bound together with a fibrinous exudate which greatly interferes with peristalsis. In some cases there is a serous exudation into the peritoneal cavity, but this is usually not marked except in the chronic tuberculous form. Pus is found especially in the perforative cases and those due to pneumococcic infection. The pus in the perforative cases has a foul fecal odor, is usually encapsulated about the appendix or site of perforation, but it may be, in the rapidly fatal cases, widely distributed throughout the general peritoneal cavity. The pus in the rare cases of pneumococcic peritonitis is very abundant, has no fecal odor, and is usually walled off and held by a single large inflammatory sac, which has a tendency to point toward the umbilicus; spontaneous perforations at this point may discharge the pus sac and result in recovery.

**Symptomatology.**—The symptomatology of peritonitis occurring during the first days or weeks of life is very insidious. The symptoms of sepsis or of intestinal irritation may be present and almost entirely mask the inflammatory process going on in the peritoneum. Then perhaps abdominal distention and tumefaction lead to a more careful physical examination of the abdomen, which may be hard, tense, and give the peculiar resistance on palpation which is more or less characteristic of inflammation of the peritoneum. In this way a diagnosis may be made before the death of the infant, as this is always a fatal disease. In many cases, however, the diagnosis is made on the post-mortem table. Peritonitis occurring in later infancy and childhood is a more open and frank disease and not necessarily a fatal one. Its onset depends largely upon the causative factors. In appendicitis a general peritonitis is announced by the sudden cessation of pain, fall in the temperature, increased rapidity of the pulse, and great prostration of the patient; these symptoms are followed, when reaction occurs, by a rise in temperature and by the appearance of the local signs of peritonitis. If the condition be due to a pneumococcus infection of the peritoneum the symptoms are sudden in their onset and general in character, with fever, chill, headache, and all the evidences of a general

pneumococcic infection such as we have in pneumonia. When these symptoms begin to subside, at the end of five or six days, abdominal pain, distention, and tumefaction call unmistakable attention to the localized inflammatory process in the peritoneum. In this form of the disease the bowels are loose and the abdomen becomes slowly distended with the fluctuating mass of pus, which may be outlined by palpation and percussion, and which usually occupies the lower middle portion of the abdomen and points to the umbilicus. In some instances, however, the pus sac may be in one or the other iliac regions, though commonly pointing to the umbilicus. The onset of gonococcus peritonitis is insidious, occurring almost always in girls, since it is commonly preceded by a gonococcus vaginitis. The character of the inflammation in these cases is for the most part mild, spreading slowly and involving, as a rule, only the pelvic or lower abdominal peritoneum. The diagnosis is made by the presence of vaginitis and by the localized pain and tumefaction in the lower abdomen and pelvis. These cases, however, as Koplik says, are not always benign, but may result in a general peritonitis ending in death.

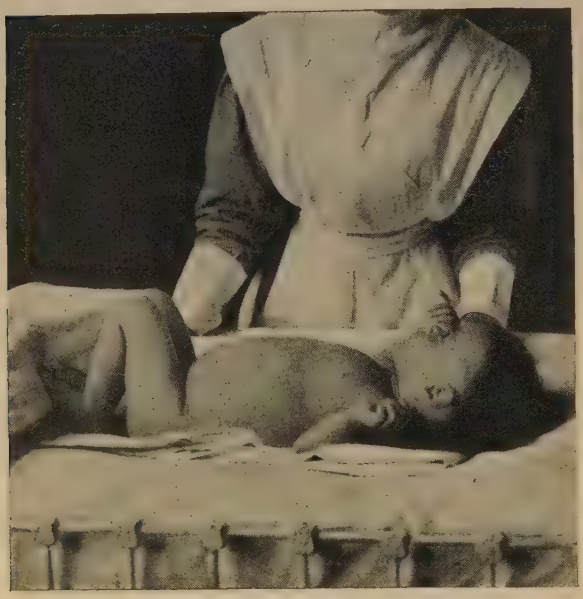


Fig. 37.—Position in Acute Peritonitis.

General Symptoms.—The pain in peritonitis is usually in the right iliac fossa or the umbilical region and from thence spreads, involving the whole abdomen. The patient lies on his back, with his legs flexed on his thighs, his abdomen distended with gas and tender to pressure. He breathes superficially and rapidly, so as not to bring into play the diaphragm or abdominal muscles. He dreads being handled or touched; his facial expression is anxious and his whole attitude is that of protecting the abdominal region from injury; his general appearance is that of serious illness; the body may be hot and dry, the extremities cold and cyanotic. Fever is almost always present, but the height of the temperature bears no relationship to the seriousness of the disease. The pulse may range from 120 to 130; it is small, weak, and increases in rapidity with the downward progress of the disease. Vomiting is commonly present and may be very

persistent; in the perforative cases its severity usually increases with the progressive involvement of the peritoneum. The vomited matter is at first food, then mucus, bile, and, in the more aggravated cases, a black coffee-ground material which may have a fecal odor. Constipation of an aggravated type is the rule, but diarrhea may occur, especially in those cases that follow the acute infections. With the progress of the disease the abdomen becomes more distended with gas, so that the liver dullness may be obliterated; general tumefaction of the whole abdominal wall becomes more and more marked, and in septic cases the fever and general symptoms are those of a septicopyemia. Leukocytosis is marked, especially in the septic cases.

Physical Examination.—The diagnosis is made by the physical examination. The tenderness may be localized or general. The abdominal resistance and induration, which are such all-important signs, may be either local or general, or may begin from a focus and spread gradually over the entire abdomen. By percussion or bimanual palpation one may localize the exudation.

**Prognosis.**—In the new-born the disease is fatal; in older infants and children the prognosis depends largely upon the character of the inflammatory process. In chronic, tuberculous peritonitis the prognosis is good. In gonococcus peritonitis a very large per cent. of the cases recover without operative interference. In pneumococcus peritonitis the prognosis is also commonly good if operative measures are resorted to at the proper time. In traumatic peritonitis, which is from its inception a purely surgical condition, the prognosis will depend largely upon the severity of the injury and early operative interference. In perforative peritonitis the prognosis has been previously discussed under Appendicitis.

**Treatment.**—In the perforative forms of this disease, as previously noted under Appendicitis, the cases are essentially surgical, and as soon as a diagnosis is made operative interference should be resorted to. In the milder traumatic forms of peritonitis, however, and in those cases which are caused by acute infections, the medical treatment may be most important. The patient should be kept absolutely quiet and not be allowed to do anything for himself that can be done by others. Cold applications should be applied to the abdomen, especially in the early stages of the disease; later, after four or five days, when the abdomen is tender to the touch, hot applications may not only be grateful but may be of value in helping nature to dispose of the inflammatory exudate within.

The dietetic treatment is most important. For the first two or three days absolute starvation is necessary, water only being allowed; after this, good whiskey or brandy, well diluted, and beef or mutton broth may be given; the subsequent dietetic treatment will depend upon the age of the child and the extent of the peritoneal inflammation, and should follow along the lines previously outlined under Chronic Indigestion. If after a few days the peritonitis is clearly demonstrated to be of such a character that perforation of the bowels is not to be feared, a saline cathartic, preferably sulphate of magnesia or Rochelle salts, should be given. The thorough

unloading of the bowels in the non-perforative cases is of the very greatest importance and materially assists in starting convalescence. In those cases, however, where appendicitis or typhoid fever either threatens or has produced perforation, cathartics are contraindicated. The use of opium may be necessary to relieve pain, but it should be used with great discretion, and when perforation has occurred it is contraindicated. The best preparation of opium is morphin and it should be given hypodermically, 1-50 of a grain for a child one year of age and 1-20 of a grain for a child six years of age, to be repeated if necessary. Enemata for unloading the lower bowel should be employed in the cases where they are not contraindicated by disease of the large intestine, such as in appendicitis. Gonococcus peritonitis is to be treated by trying to cure the causative vaginitis (a very difficult matter) and by general tonic treatment, including proper food, iron, cod-liver oil and fresh air, and by the local treatment above noted. Pneumococcus peritonitis is to be treated by general sustaining measures, such as are used in pneumonia, and later by operative measures for getting rid of the pus in the abdominal cavity.

## ASCITES

Ascites, or the accumulation of serum in the peritoneal cavity, has its origin in children commonly in a tuberculous inflammation of the peritoneum. It may also be caused by an atrophic cirrhosis of the liver, tumors, enlarged lymph nodes which obstruct the portal circulation, diseases of the heart, producing a failure in the general circulation, Bright's disease, and severe anemia.

**Differential Diagnosis.**—Ascites from cardiac weakness is always associated with swelling of the legs and with unmistakable symptoms pointing to disease of either the cardiac valves or muscles. Ascites from Bright's disease is associated with a general anasarca and the urine findings of that disease. Ascites from grave forms of anemia is always associated with a cachexia, and a blood examination establishes the diagnosis. Ascites from local disturbances of the portal circulation may be confused with tuberculous peritonitis. Cirrhosis of the liver is rare in children and is commonly of syphilitic origin. Other evidences of syphilis and the absence of the physical signs of peritonitis usually suffice to make the differentiation; if not, after the abdominal fluid has been removed by tapping, a contracted and nodular liver may be demonstrated, or, failing in this, the negative tuberculin skin reactions may throw light on the subject. Tumors and enlarged lymph nodes (tuberculous) of sufficient size to produce ascites by obstructing the portal circulation can, as a rule, be demonstrated by palpation after removal of the serum. In the presence of a marked ascites in children the physician should remember that tuberculosis is the common cause of this condition, and that often the abdominal cavity in this disease may be distended with serum without any very acute symptoms; there may

be very little tenderness and induration of the abdominal wall and the condition may have developed very insidiously. For these reasons ascites due to a low grade of chronic tuberculosis is very commonly suspected to be due to other causes. The importance, therefore, of making a thorough examination for every sign or symptom of tuberculosis cannot be exaggerated.

## CHAPTER XXVIII

### THE RECTUM AND ANUS

### MALFORMATIONS OF RECTUM AND ANUS

Complete or partial congenital occlusion of the rectum or anus may occur. *Atresia of the anus* may result from the failure to absorb the skin covering, which normally guards the rectum during intrauterine life. This condition may usually be made out a few hours after birth by the bulging of the rectum beneath the skin, and a slight exploratory incision discovers the rectum and cures the deformity. *Atresia of the rectum* is a more seri-

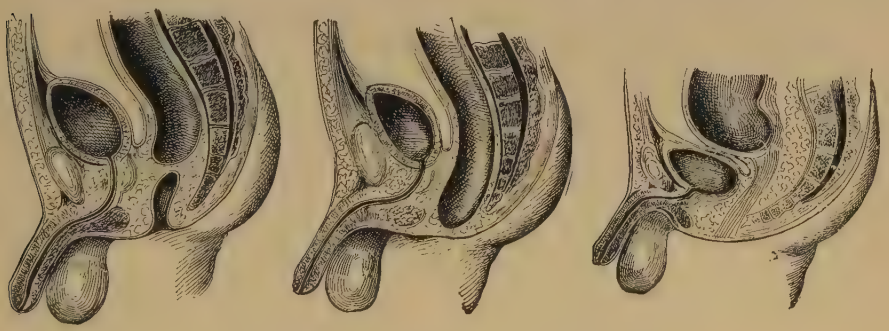


FIG. 38.—MALFORMATIONS OF THE RECTUM.

ous matter; in this condition the occlusion of the rectum is commonly located two or three inches from the anus. In some instances the anal end of the rectum below the occlusion is patulous and continuous with a patulous anus; in such cases the exploring finger introduced through the anus into the rectum may readily reach the septum and direct the trochar which relieves the obstruction by puncturing this septum. In another group of cases there is *congenital absence of the rectum* below the point of occlusion. The blind sac of the rectum, some inches from the anus, is connected with it by an impervious cord, which represents the undeveloped rectum. In some of these cases the contents of the rectal pouch find their way by fistulous tracts through the peritoneum or into the bladder, vagina, or urethra. In those cases where occlusion is complete immediate surgical measures are necessary. In other cases, however, where the fistulous tracts are wide enough to serve the temporary purpose of emptying the

rectal pouch, a surgical operation may be postponed for a short time until the infant is stronger.

## POLYPUS OF THE RECTUM

Polypus of the rectum is not infrequent in children; the tumor is attached by a pedicle to the rectal wall and is usually single, but occasionally more than one tumor is present. It may exist for a long time without presenting itself at the rectum and during this time there may be more or less rectal irritation, with tenesmus and blood and mucus in the stools. The diagnosis may be made by introducing the finger into the rectum; in this way the tumor may very readily be outlined and differentiated from hemorrhoids and intussusception. A rectal examination should be made in all cases in which there is an unexplained hemorrhage from the rectum.

**Treatment.**—Rectal polypi may easily be removed by twisting the pedicle with forceps or with a wire snare. Following their removal, the rectal irritation rapidly subsides and the growth does not commonly recur.

## PROLAPSE OF THE RECTUM

This condition is usually seen in children under six months of age; the almost straight rectum, with its weak attachments at this period of life, predisposes to prolapse. It occurs more commonly in malnourished children suffering from diarrhea and constipation. Rectal, vesicle, or genital irritation produced by thread-worms, fecal concretions, rectal polypi, vesicle calculi, cystitis, urethritis, and phimosis may be factors in producing it. In mild cases the mucous membrane of the anus may be but slightly everted when the bowels are moved and the blood and mucus may appear upon the napkin. In more severe cases the rectal wall may be prolapsed, forming a dark-red corrugated tumor two or three inches long, which bleeds readily on manipulation; at the end of this tumor a depression marks the anal opening. The prolapsed rectum can, as a rule, easily be replaced by gentle pressure, the prolapse recurring again when the bowels are moved. In rare instances the tumor remains down and cannot be replaced by simple manipulation; in such cases the parts may bleed, become much swollen and inflamed, and strangulation and ulceration of the mucosa may occur.

**Treatment.**—The reduction of the tumor mass is commonly easily accomplished by manipulating it gently upward through a cold, moist towel; if difficulty is experienced in reducing the tumor the child should be placed in bed, stomach downward, and cold compresses applied to the part. Following these applications, the tumor may be easily reduced; if not, a few inhalations of chloroform will relieve the contracted muscles and make the reduction of the prolapsed rectum an easy matter. The rectum having been returned to its normal position, the object of all subsequent treatment is to keep it there. In order to accomplish this it is

advisable to keep the child in bed, or at least in a reclining posture, until all local irritation of the rectal mucous membrane has been removed. Pinworms and colitis, if they exist, are to receive prompt attention. If phimosis be present the infant is to be circumcised, as the reflex irritation from the genital organs may be an important factor in producing subsequent attacks of prolapse. The rectum may be retained in position by strapping the buttocks with adhesive plaster when the child is on its feet and by having the child lie down before every movement of its bowels. Following the evacuation of the bowels, a large injection of cold, normal salt solution should be given; these cold injections are of great value in the treatment. In still more severe cases the thermo-cautery may be used on the prolapsed mucous membrane, making a number of linear cicatrices in a longitudinal direction. In rare instances amputation of the tumor may be necessary; this operation is usually attended with success.

## FISSURE OF THE ANUS

This condition is not uncommon; it is produced by constipation; the passage of large, hard, fecal masses stretches and tears the mucous membrane about the anus. Infection may follow this injury and a small fissured ulcer may form, coated with pus and mucus and imbedded in the folds of the mucous membrane, which is more tightly grasped by the sphincter ani, because of the irritation produced by the fissure. Fischl says that fissures of the anus occur almost exclusively on the posterior rectal wall, and that they can be seen only by placing the child upon its back, with its pelvis elevated and legs widely separated and flexed on the trunk; in this position, with fingers on either side of the anus, pressing it apart, the fissure is exposed. The condition may be complicated or even produced by the presence of pin worms, the parts being torn by scratching. These ulcers are extremely painful when they are touched or when the rectum is manipulated in any way. Defecation is resisted by the child and, when it can no longer be postponed, causes great pain. The chronic constipation which is so commonly the cause of this condition is still further aggravated by its presence, the powerfully contracted sphincter resisting the passage of fecal masses. There is little tendency to spontaneous recovery. So great is the pain produced by defecation in some of these cases that retention of urine occurs as a result of the child's dread of bringing into action the muscles of the bladder, which are so closely associated physiologically with the muscles of the rectum.

**Treatment.**—The constipation should be relieved by laxative medicines, such as compound licorice powder for children and milk of magnesia for infants. In mild cases the fissure should be treated daily by carefully cleansing with a cotton-wrapped probe and then touching it with a 2-per cent. solution of cocain muriate, following this by the application of a 10-per cent. solution of nitrate of silver. Under this treatment mild cases recover. The strength of the silver nitrate solution must be regulated by

the pain and discomfort which follow its use. More severe cases are to be treated surgically; these usually get well following thorough dilatation and stretching of the sphincter under complete anesthesia.

## SPASM OF THE ANUS

This condition is commonly due to fissure; it may occur in neurotic children from causes which produce irritation of the rectal mucous membrane; it produces constipation, tenesmus, and pain on defecation. For the relief of this condition mild laxatives are indicated; warm olive oil injected into the rectum may be of value. If these means fail, forcible dilatation of the sphincter muscle should be resorted to.

## PROCTITIS

Proctitis, or inflammation of the rectum, may exist unassociated with catarrhal conditions of other portions of the intestinal mucous membrane. It may be produced by thread worms, by the frequent use of glycerin, soap, and other irritating suppositories, by the careless use of the thermometer, by rectal tubes used in giving enemata, or it may be a complication of vulvovaginitis. It is characterized by tenesmus, constipation, painful defecation, and a discharge of pus and blood associated with tenesmus. It may be relieved by mild cathartics and rectal injections of saline solutions or olive oil. Occasionally mild astringent solutions of one-half per cent. of nitrate of silver are indicated.

# SECTION V

## *NUTRITIONAL DISORDERS*

## CHAPTER XXIX

### RICKETS

### (*Rachitis*)

Rickets is a chronic disease characterized by nutritional disorders, and consequent lack of development and perverted function on the part of nearly every organ and tissue of the body. It affects chiefly the bones, nervous system, muscles, mucous membranes, ligaments, and blood, and is believed to be largely a disturbance of calcium metabolism.

**Etiology.**—Rickets is a disease of infancy; more than 80 per cent. of the cases occur under two years of age; it rarely begins during the first three months and is unusual after the third year. It is a very common and widespread disease; in our largest cities perhaps 90 per cent. of the infants of the poor show some signs of rickets. It is, however, much more common in cities than it is in the country, because a greater percentage of country children are breast-fed, live under better hygienic conditions, spend a greater portion of their time in the open air, and are altogether better fed. Heredity is an important factor.

Rickets occurs more commonly in cold than in warm climates. This is due not so much to climate as it is to the conditions of life which cold climates force upon a poverty-stricken population. Impure air and lack of sunshine are very important causative factors which act largely through the unfavorable influence they exert on the child's digestive capacity.

*Faulty feeding* is the most important cause of rickets. This disease rarely occurs in breast-fed babies, unless lactation is prolonged or the mother's milk, by reason of her vocation or ill health, furnishes insufficient nutrition to the rapidly growing infant. It is, however, very common among artificially fed infants who are insufficiently nourished on improper food formulas. A marked excess of carbohydrates and a notable deficiency in fat, protein and salts are the special dietetic errors most closely related to the etiology of rickets. This is the reason condensed milk, Nestlé's food, malted milk, and other proprietary foods which do not require fresh milk in their preparation, are such potent factors in producing rickets.

It is a well-founded belief that a deficiency of fat in the infant's food is the most important of these dietetic errors and is most commonly related to the etiology of rickets. The absence of fat interferes with the proper assimilation of lime and phosphorus and this, perhaps, explains the potency of fat starvation as an etiological factor of this disease. The prevalence of rickets among the negroes and Italians of our large cities is not due to racial or hereditary influences, but is purely a question of bad hygiene and improper food.

**Pathology and Morbid Anatomy.**—At the junction of epiphysis and diaphysis the ribs and long bones are enlarged by a proliferation of poorly constructed vascular cartilaginous and bony tissue between the epiphyseal

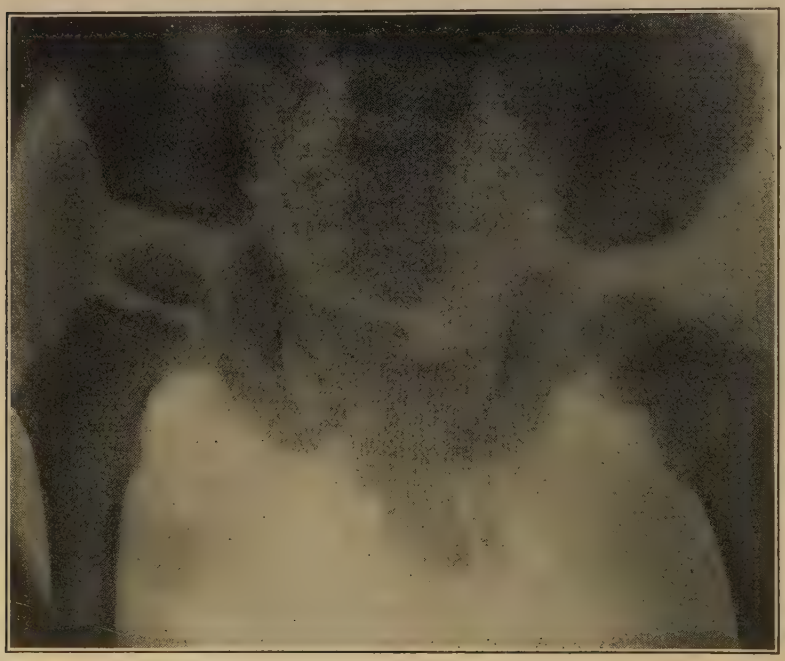


Fig. 39.—Bony Deformities in Rickets. (A. Freiberg.)

cartilage and the cancellous portion of the diaphysis. Bony structures everywhere, and especially the long bones, are more vascular and cancellous than normal, and there is increased resorption of bone; the subperiosteal calcification and formation of new bony tissue is interfered with. Such bones contain only one-third instead of the two-thirds mineral matter which normal bones contain. These changes produce an increased flexibility of the bones which predisposes the infant to the deformities so characteristic of rickets.

Muscular tissue is everywhere poorly formed, and the muscular fibers are infiltrated with fat and may show partial fatty degeneration. The muscles are poorly nourished, weak, flabby, easily stretched, and offer an

insufficient support to the viscera and bones which they cover. The ligaments are weak, flabby, easily stretched, and do not properly sustain the bony structures.

The spleen is notably enlarged, it is anemic, its Malpighian bodies are atrophied and its function as a blood-forming organ is interfered with. The lymph nodes are slightly enlarged. The liver is somewhat increased in size and displaced downward.

**Symptomatology.**—Rickets is a chronic malnutrition affecting every part of the body. The bony lesions are the most pronounced, the most characteristic, and the most easily recognized, but they are not the earliest nor are they the most important signs of this disease. A general failure of nutrition, manifested by the following symptom group, presents the syndrome upon which the diagnosis is made; retardation in physical development; muscular weakness; inability to sit erect, to hold the head up, or to use the legs in a normal manner; flabby and undeveloped muscles; marked anemia; general nervous irritability; scarcity of hair on the back of the head due to restlessness and head sweating when asleep; tendency to fever from slight causes; late teething; a large, square, flat head with open fontanels; a chest contracted above and constricted transversely at the diaphragmatic attachment; flaring ribs below the diaphragm, turned upward upon a markedly distended abdomen; a rickety rosary of bead-like prominences at the junction of the ribs with the costal cartilages; knob-like prominences of the bones just above the ankles and wrists; constipation; a tendency to gastrointestinal disturbances and bronchial catarrh; large belly; enlarged spleen and a liver protruding below the costal margin. In the more severe forms of rickets the above symptoms are much exaggerated and the deformities which result from diseased viscera, weak ligaments, ill-developed muscles, and flexible bones, are very great.

Fig. 40.—Rickets.

General Appearance.—Rachitic babies in the early stages may be fat and flabby, so that to the prejudiced eye of the mother they may appear normal, while to the practiced eye of the physician they present at a glance the characteristic signs of rickets. As the disease advances, however, they

become emaciated and then the thin, old face, large, square head, resting on a narrow, contracted, deformed chest, the large, distended abdomen, the crooked back, the thin, bent, and deformed arms and legs present the characteristic picture of advanced rickets with which even the laity are familiar.

General Symptoms.—Head sweating when the infant sleeps is one of the early significant symptoms and should lead to a search for other signs of rickets. Delayed and difficult dentition is an almost constant accompaniment of even the mild forms of rickets. In a normal infant a tooth may come through with little or no constitutional disturbance, but in a rachitic child it commonly produces fever, sleeplessness, general nervous irritability, and some slight gastrointestinal disturbance. In fact, the severity of these symptoms, on the cutting of a tooth, may be an important indication not only of the presence of rickets, but of the severity of the nutritional disturbances which it has produced in the nervous system. The teeth of the rachitic infant are poorly formed and decay early.

*Nervous symptoms* are of great value in the early diagnosis of rickets. Rachitic children are fretful and nervous, poor sleepers, toss restlessly in their sleep, and, as a result, very commonly have an occipital baldness. Reflex and toxic agents have a highly exaggerated influence on their nervous systems; slight reflex and toxic factors in the intestine and elsewhere produce high fever and convulsive symptoms. The predisposition of the rachitic infant to fever and convulsive disorders occurs early and may be an aid to an early diagnosis; convulsions or fever occurring in an infant from a trivial or from no apparent exciting cause should lead to a careful search for other signs of rickets. Laryngismus stridulus and tetany are nervous syndromes, very closely associated with the more advanced and severe types of rickets. Spasmophilia, or exaggerated peripheral nerve excitability, is one of the most characteristic phenomena seen in rachitic children.

*Gastrointestinal disorders* occur very commonly and very early in most cases of rickets; in this disease there is a predisposition to catarrhal diseases which may be especially marked on the part of the gastrointestinal tract. In some instances the gastrointestinal disturbances precede the rickets and may be considered as causative factors. The pot-belly of rickets, which develops early, is an important symptom and is associated with frequent attacks of indigestion and intestinal fermentation; in this condition the abdominal muscles are relaxed, flabby, and greatly distended by the flatulent intestines. The enlarged spleen and the downward displacement of the liver can easily be made out by palpation and percussion.

*Hernias,* both inguinal and umbilical, are very frequently seen in rachitic infants; in fact, rickets is the most common predisposing cause of infantile hernias. The recti muscles are sometimes separated as much as an inch by abdominal distention.

*Weak ligaments* and muscles are always present and are largely responsible for the helplessness or physical backwardness of the rachitic infant; the curvature of the spine and the tardiness of the infant in sitting, stand-

ing, walking, and in making all the complicated muscular movements are largely due to this cause.

*Bony deformities* are the most characteristic and easily recognized signs of rickets; some of these occur early in the disease. The beading of the ribs, or rickety rosary, the horizontal depression of the ribs at the diaphragmatic attachment and their flaring upwards below this line, the marked enlargement of the long bones just above the wrist and ankle may be counted among the bony changes which are of great value in the early diagnosis of rickets. As the disease advances the bony deformities are more exaggerated; the soft vertebræ, with their relaxed ligaments and weak muscular support, result in gradually increasing curvatures of the spine; the posterior curvature (kyphosis) is the most characteristic and commonly involves all the lower portion of the spine below the mid-dorsal region; extensive lateral curvatures are also common; rotary curvatures are also noted. Rachitic curvatures are not as sharp and angular as those due to tuberculous disease and they are not, especially in their earlier stages, fixed; they will, as a rule, entirely disappear when proper pressure and extension is applied. The clavicle may be curved upward and forward in its inner third. In severe cases the pelvis is permanently deformed, small, and especially contracted in its anteroposterior diameter. The arms and legs may be greatly deformed by the curving and twisting of long bones; the humerus may be curved outward and the natural outward curve of the radius and ulna may be exaggerated. In the lower extremities the most common deformity is an outward curvature of the lower third of the tibia, producing "bow legs"; we may also, much less frequently, have "knock-knees," which

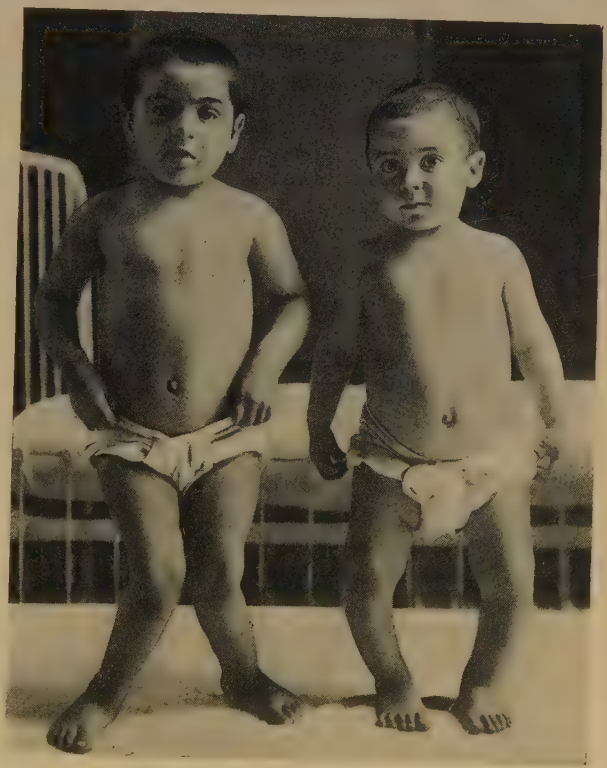


Fig. 41.—Knock-knees and Bow-legs Due to Rickets in a Sister and Brother, Aged Five and Three.

Holt says "are more common in females and are believed to be due to an overgrowth of the inner condyles of the femur." The cranium presents some of the most important characteristic bony deformities. The head is larger than normal and the anterior fontanel is much delayed in closing; at one year of age it may measure from 1½ to 2½ inches in both diameters, while at this time it should not measure more than ½ or 1 inch; it may remain open to the end of the third year. The top of the head presents a flattened, square appearance due to thickening of parietal and frontal eminences. In young infants soft and yielding spots, due to thinning of the bone in the parietal and occipital regions, are found; these patches may be from ½ to 1 inch in diameter. This condition, known as craniotabes, is not more characteristic of rickets than it is of syphilis; it occurs in both conditions and is very commonly associated with the syndrome of laryngismus stridulus.

Blood Changes.—According to Morse, a number of forms of anemia may occur, the red cells are slightly reduced, and the hemoglobin very much so, from 30 to 40 per cent. The specific gravity is reduced and leukocytosis is present when the spleen is markedly enlarged.

The mucous membranes of rachitic infants are especially prone to catarrhal inflammations from slight causes. Gastrointestinal catarrh, coryza, pharyngitis, laryngitis, bronchitis, and pneumonia are common complications of rickets.

Course.—Rickets is a chronic disease and its duration will depend on its severity and the character of the treatment instituted. In mild cases, under proper treatment, it may be cured in three or four months. In severe cases the active symptoms may last from eighteen months to two years. Many of the rachitic deformities are permanent.

Congenital rickets is a rare disease. It does, however, occur in utero; infants are born with craniotabes, the rickety rosary, and other characteristic bony changes; this form of the disease is especially rare in America.

Late rickets, occurring in children from six to ten years of age, is very uncommon. In these cases the bony deformities develop rapidly. I have seen but one case, a girl seven years of age in whom there rapidly developed a softening and bending of the bones of the legs and other characteristic symptoms. Braces were applied and treatment instituted. The child recovered after ten or twelve months' treatment.

**Diagnosis.**—The diagnosis of well-marked rickets is easily made and attention will only be directed to the fact that it should be made in most cases much earlier than it is. If nervous, irritable children, who have been fed on a food rich in starch and poor in fat and protein, show general lack of development, late teething, open fontanels, muscular weakness, and head sweating during sleep, a diagnosis of rickets should be made without waiting for the disease to proclaim itself by its more characteristic symptoms.

**Prognosis.**—The prognosis in cases of mild rickets is very good; the disease, in this stage, contributes little or nothing to the mortality list.

Severe rickets, however, is a very grave disease and contributes largely to the death list in our large cities; not that rickets itself is so dangerous, but it reduces the vitality of the infant to such a low ebb that it readily succumbs to catarrhal diseases of the gastrointestinal and respiratory tracts, as well as to convulsive disorders which are such common complications of rickets.

**Treatment.**—Prophylaxis.—Since rickets is such a common disorder in artificially fed infants, and since the disease can, as a rule, be prevented by careful feeding and proper hygiene, the physician should have in mind the prophylactic treatment of rickets in the feeding of every baby that comes under his care. In breast-fed babies the prophylactic treatment of infants is not, as a rule, difficult; it is only necessary to be sure that they have a sufficient amount of good breast milk, and, where there is any doubt upon this question, to supplement the breast feedings with properly modified fresh cow's milk, according to the rules outlined under Mixed Feeding. If the breast milk is sufficient and of proper quality, it is safest to continue to feed the baby exclusively on breast milk for nine months and then to supplement the breast feedings with fresh, clean cow's milk. In artificially fed infants, however, the problem is a very different one. It is easy enough to write out a milk formula for an infant with normal digestive capacity which will furnish it with the proper number of calories and with the proper percentages of fat, protein, salts, and carbohydrates to prevent the development of rickets. But there are other infants, born of delicate parentage, suffering from mild forms of glandular tuberculosis or other constitutional diseases; premature infants and infants who because of gastrointestinal disorders have a feeble digestive capacity; these are the cases that give us trouble and that force us to a compromise which results in a food formula which the infant can digest, but which may fail to fully supply its nutritional demands. In these cases it is difficult for even the most experienced physician to carry the infant through the first year without the development of at least a mild form of rickets, unless a wet-nurse is employed.

Curative Treatment.—From what has been said it is clear that the important factor in the treatment of rickets is the *diet*. The disease has developed upon a food formula which failed to meet nutritional demands, and this formula is commonly rich in carbohydrates and poor in fat, protein, and salts, such as we find in condensed milk, Nestlé's food, malted milk, and other proprietary foods which do not require fresh cow's milk in their preparation. It follows, therefore, that it is absolutely necessary to success in the treatment of rickets, that the food upon which the infant has developed the disease should be either radically changed or greatly modified. A mistake, however, which is very common and which, as a rule, is fraught with disastrous results is a too radical change in the diet. Many rachitic infants, when they come under the observation of the physician, have for a long time been fed upon one of the easily digested proprietary foods and have, therefore, such feeble digestive capacity that they cannot at once be placed upon cow's milk. If such a radical change is attempted

in these cases it is probable that gastrointestinal disturbances will be added to the other rachitic symptoms. It is necessary, therefore, in changing the food, especially of infants with advanced rickets, to make haste slowly. One may, perhaps, in these cases begin by adding skimmed raw milk to the food the infant is already taking and gradually increasing the milk so as to develop the digestive capacity. In this way the original proprietary food mixture may be gradually replaced by skimmed milk and later by whole milk, until a food formula is reached which in fat and proteins will serve the nutritional demands of the infant. It may be necessary in some instances to use peptonized milk to gradually replace the proprietary food and then later to gradually diminish the peptonization until the baby is on a suitable raw-milk formula. If the infant has developed rickets on sterilized milk this food must be replaced by raw milk. By this gradual process of eliminating an unsuitable food the child may be placed upon a proper food mixture without producing digestive disturbances. The object in every instance being to increase the proteins, fat, and salts and diminish the carbohydrates, but in doing this great care must be taken to prevent overfeeding; the value of the food mixtures in calories should at no time greatly exceed the nutritional demands of the infant. It is also wise to feed all rachitic infants at a four-hour interval; more frequent feedings are generally fraught with disastrous results.

In the second year of life soft-boiled eggs are a very important adjunct to the dietetic treatment; the yolk of the egg furnishing the fat and the white the albumin. Meat juice and beef peptonoids may also be of value in those cases where the idiosyncrasies of the child make it impossible to put it upon a proper milk formula. Butter is a palatable and easily digested fat which may be given on bread or in a cereal with milk. Raw or partially cooked, scraped beef is a food of great value when it can be properly cared for by the digestive organs of the child.

*Fresh air* and *sunshine* are curative measures of the greatest importance. There can be no doubt that the fresh air and sunshine of the country, under suitable climatic conditions, are of themselves direct curative agents, but their greatest importance lies in the fact that they very materially increase the digestive capacity of the infant for fat and proteins, and thereby hasten the time when it can be placed upon a proper diet.

*Cod-liver oil* is, next to a suitable diet, the most valuable remedy we have in the treatment of rickets, and the excellent results which come from its use seem to indicate that its great value does not altogether lie in the fact that it is an easily assimilated fat. The administration of cod-liver oil, however, should not be begun, in advanced cases, until we have partially solved the food problem as outlined above; but in mild cases it should be begun at once and in severe cases as soon as the physician believes that the digestive capacity of the infant will permit of its administration. As a rule, cod-liver oil is well tolerated by rachitic infants; it should be administered over a long period of time in connection with the dietetic treatment. The form in which cod-liver oil is given will depend upon the individual

idiosyncrasies of the infant; it should be discontinued if it produces lack of appetite or gastrointestinal disturbances; it is perhaps best given combined with one of the malt extracts. These preparations are palatable and, as a rule, improve the digestive capacity of the infant. In individual instances, however, it may be better to give the pure oil or one of the palatable emulsions. For an infant six months old the dose may be from 15 to 20 minims three times a day, and for an infant one year of age twice this amount. *Phosphorus,* so highly recommended by Kassowitz and Jacobi, is of value in the treatment of rickets; the dose should be from 1/200 to 1/250 of a grain. It may be given in the form of Thompson's solution of phosphorus, which may be mixed with whatever form of cod-liver oil preparation the infant is taking. *Iron,* in the form of the saccharated carbonate or some other easily assimilated preparation, is of value where there is marked anemia with an enlargement of the spleen and lymph nodes. It is, however, advisable to get the infant well started on a proper food formula, so as to have its convalescence well under way, before beginning the administration of iron.

*Salt baths* have been recommended and are perhaps of some value during the acute stage of the disease.

*Oil inunctions* are believed to be of value throughout the whole course of the disease. Mild general massage, followed by inunctions of anhydrous lanolin, are of value in improving nutritional conditions. This treatment gives mild exercise to the wasted muscles and joints and also gives the infant a certain amount of fat, which is taken up by the lymphatics and blood vessels of the skin.

Prevention of Deformities.—The prevention of deformities is one of the most important duties which the physician has to observe in the treatment of acute rickets. During the active stage of the disease, while the bones are soft and flexible, it is most important that the infant should be prevented from assuming positions that will result in bony deformities. It should lie upon its back on a smooth mattress, and, when it is handled, care should be taken that long-continued pressure in any one position may not result in the curvature of bones. It should be discouraged from walking, crawling, and sitting alone until the treatment has been continued long enough to overcome the softness and flexibility of the bones. It may in some instances be necessary, if the child is slow in its recovery, to resort to braces and other supports to prevent curvatures of the spine and of the bones of the leg.

Treatment of Rachitic Deformities.—There is a tendency on the part of nature to gradually overcome the deformities which result from the contraction of the ribs, the curvature of the spine, and the distended abdomen. She may be assisted in her laudable purpose by subjecting the child to systematic gymnastic exercise under the guidance of a competent instructor. The surgical treatment of old rachitic deformities, especially of the long bones, is of great value, but this is a subject which belongs to the field of orthopedic surgery.

## CHAPTER XXX

### INFANTILE SCURVY

*(Scorbutus)*

**Definition.**—Scurvy is a chronic nutritional disorder due to a prolonged absence or diminution of certain food constituents which are absolutely necessary to normal metabolic processes; the exact nature of this food deficiency is not altogether clear. The medical world inclines to believe with Barlow, Northrup, Crandall, and others, that infantile scurvy is the same disease as scorbutus in the adult; the clinical picture being modified, as it is in so many other diseases, by the somewhat different chemical and biological problems found in the immature and rapidly developing organism of the infant. Rickets and scurvy are so commonly associated in the infant that for many years there was great confusion in their differential diagnosis, it being commonly believed that scurvy in the infant was a manifestation of rickets. In recent years, however, the well-recognized hemorrhagic tendency of infantile scurvy and its general resemblance to scorbutus in the adult have led to the very general belief that it is an independent affection commonly associated with, but not otherwise related to, rickets either in its etiology, pathology, or treatment.

**Etiology.**—Infantile scurvy is for the most part a disease of the first and second years of life; the great majority of cases occur between the sixth and the eighteenth month; it rarely begins before the third month or after the second year. It occurs more frequently in infants of the middle and upper classes, because they are not uncommonly fed on a food which at some time or other in its preparation has been subjected to superheating; in this it differs markedly from rickets, which is more common among the poor because of the ill-balanced food formulas upon which these infants are fed.

**Diet.**—Some dietetic error is the all-important cause of the development of scurvy. The report of the American Pediatric Society's Collective Investigation of Infantile Scurvy in 1898 showed that of 379 cases the food upon which the disease developed was as follows:

| | | | | |
|---|---|---|---|---|
| Breast-milk | in | 12 | cases, alone in | 10 |
| Raw cow's milk | in | 5 | cases, alone in | 4 |
| Pasteurized milk | in | 20 | cases, alone in | 16 |
| Condensed milk | in | 60 | cases, alone in | 32 |
| Sterilized milk | in | 107 | cases, alone in | 68 |
| Proprietary infant-foods | in | 214 | cases. | |

This report shows that scurvy most commonly develops on the proprietary foods, sterilized milk, condensed milk and pasteurized milk and that it very uncommonly develops on raw cow's milk and breast milk. In most of the cases in which scurvy has developed on breast milk, the milk is either

defective in composition, as shown by chemical analysis, or exclusive breast feeding has been continued for too long a time.

In studying the dietetic causes of scurvy one is led to the conclusion that the fault must lie chiefly in the absence of some fresh principle in the food, which is either destroyed, chemically changed, or rendered less digestible or assimilable by heat. It is evident that heat may act by destroying active biological properties of the food which are necessary in infantile metabolism, or it may act by producing changes in the acids and salts of fresh foods; these acids and salts being in part separated from their protein combinations by heat and their biological and chemical value thereby impaired in the body metabolism. Whether or not this be the explanation, the fact remains that food that has been biologically killed by heat, as in the sterilization of milk or in the preparation of proprietary foods, is responsible for over 90 per cent. of the cases of infantile scurvy. The character of the food formula, which is all-important in the etiology of rickets, has little to do with the production of scurvy, and the fact that the proprietary foods which are such a prolific cause of scurvy in infants are also ill-balanced in the percentages of their important ingredients, explains why scurvy and rickets are so commonly associated in the same infant. A food that contains too little fat, protein, and salts may produce rickets, while, on the other hand, as a result of the changes which heat has produced in it, it may produce scurvy.

A very small percentage of the cases occur in children fed on breast milk and on raw cow's milk. That these unusual cases cannot in every instance be explained by a chemical analysis of the milk does not militate against the fact that food that has been changed by heat is the all-important cause of this disease. When scurvy occurs in an infant fed on breast milk or raw cow's milk the rational conclusion is that this particular milk has suffered some important chemical or biological change, even though the science of chemistry may not be able to reveal its nature.

**Morbid Anatomy.**—The characteristic changes are produced by hemorrhages which may be very widespread; the most notable are the subperiosteal hemorrhages of the long bones, which may be very extensive along their shafts and at their epiphyseal junction. The diaphyses and epiphyses of these bones may separate, causing marked deformities. Hemorrhages may also occur in the medullary canals and characteristic changes occur in the bone marrow, which becomes poor in cells and blood vessels. The muscles, the pleura, the pericardium and peritoneum may be the sites of small hemorrhages, and the gums are spongy and hemorrhagic. In a large percentage of the cases the minute changes occurring in bones are similar to those seen in rickets, but these changes are caused by the rickets, which is so commonly associated with scurvy, and do not belong properly to the pathology of this disease.

**Symptomatology.**—Tenderness of the legs and sometimes of other portions of the body is one of the earliest and most characteristic symptoms; this is manifested by the infant crying when it is handled and by its re-

maining quiet when it is allowed to rest in its bed. The importance of this sign is emphasized by the fact that the natural instincts of the infant are here reversed; if an infant, contrary to all the instincts of its nature, cries and frets when it is taken up and fondled by its mother and becomes quiet again when it is replaced in bed, this can only be explained by the fact that the handling causes pain. This leads to the suspicion that the child has been injured, and an investigation on the part of the mother often confirms her in this belief, since she finds that the child, even when lying in its bed, will cry with pain when some particular portion of the body is moved. A further investigation may develop the fact that the legs are tender to the touch and are swollen about the knees and ankles. These swellings are commonly fusiform in shape and are due to the subperiosteal hemorrhages, which may extend from the ankle to the knee, and, being more marked near these joints, may in severe cases cause a separation of the epiphyses and diaphyses. The soreness and tenderness produced by these swellings cause the infant to hold its legs as motionless as possible, thus producing the pseudo-paralysis of scurvy. This false paralysis results from the tense condition of the muscles which holds the legs immovable and is perhaps the result of reflex rather than of voluntary action on the part of the infant; this condition of immobility is even greater when the spinal column is involved. The severity of these symptoms grows apace as the disease advances, until the infant is in a pitifully helpless condition, screaming with pain at the slightest movement or crying out with fear when it is approached by the physician. In these severe cases the sternum may be separated from the ribs and sunken backward, producing a characteristic deformity.

Hemorrhage into the gums is an early and characteristic symptom. The gums may be purple and swollen and when teeth are present hemorrhages are more frequent; they may bleed when touched and there may be evidences of spontaneous petechial hemorrhages in the vault of the pharynx and other parts of the mouth. Hemorrhages may occur from other mucous membranes, as is evidenced by the fact that blood is occasionally found in the feces and in the urine. They may also occur in the subcutaneous tissues about the joints and other parts of the body, as well as in the conjunctiva and orbit. In this latter position they may produce exophthalmos.

Enterocolitis may be present in severe cases. The child is irritable, anemic, and suffers from general malnutrition; in advanced cases the anemia and malnutrition are very marked. The blood findings may be those of an ordinary secondary anemia or they may be of the chlorotic type. The urine not uncommonly contains albumin and casts. In advanced cases fever is usually present, but it is inconstant and irregular in type and is perhaps due to intestinal and other complications.

**Diagnosis.**—The crying of the infant on being taken up, the swelling and tenderness of the joints, the hemorrhagic condition of the gums, and the history of the child having been fed upon a food which may develop scurvy, are commonly sufficient to make the diagnosis. In making a differ-

ential diagnosis, however, the physician should remember that rheumatism presenting the above symptoms very rarely, in fact almost never, occurs in infancy, but if rheumatism, osteomyelitis, or periostitis be suspected in an infant presenting the above symptoms, the differential diagnosis of these diseases from scurvy can be readily made by the dietetic treatment subsequently outlined.

**Prognosis.**—The prognosis, when the disease is recognized early, is good. In neglected cases which have come into the hands of the physician too late to respond to treatment, death may occur from malnutrition, enterocolitis, pneumonia, or hemorrhage.

**Treatment.**—PROPHYLACTIC TREATMENT is of the very greatest importance. In many instances the physician, especially in dispensary practice, is forced to superintend the feeding of an infant during the hot months of summer, where the conditions are such that he must use foods, the long continuance of which may produce scurvy. When these sterilized and cooked foods are given for any length of time it is certainly the part of wisdom to direct that from time to time these infants be given a certain amount of orange juice. This will effectually prevent the development of scurvy.

The CURATIVE TREATMENT of scurvy is almost as simple as the prophylactic treatment and consists in giving the infant some kind of fresh or uncooked food, either with or as a substitute for the food which it has been taking. Fresh fruit juice is a specific for scurvy. Orange juice is generally used, because it can be obtained at any season of the year, and because it more commonly agrees with the infantile digestive organs than other fruit juices. One may begin by giving a teaspoonful of orange juice, slightly sweetened, if necessary, five or six times a day. An existing gastroenteritis does not contraindicate the use of orange or other fruit juices. Under this therapeutic measure the pain and tenderness about the joints may disappear in four or five days, and a cure may confidently be expected within two or three weeks. As the child improves and the gastrointestinal condition becomes normal, the amount of orange juice may be increased to six tablespoonfuls in the twenty-four hours. It may be advisable to give fresh beef juice in connection with the fruit juices in the beginning of the treatment. Later, potatoes that have been steamed and mashed and the juice of fresh vegetables may be given with advantage. The mistake is very commonly made in advanced scurvy of immediately attempting to substitute a diet of fresh cow's milk for the proprietary food upon which the infant has been living. There can be no question as to the advisability of such a procedure provided the infant's digestive capacity is equal to the change. But in most instances this change has to be brought about very gradually and should not be begun until the infant has commenced to respond to the specific treatment above noted; the change to fresh cow's milk is then brought about as rapidly as the infant's digestive capacity will permit. The raw cow's milk itself, like the fruit juices, has a curative influence and is also necessary to remove the malnutrition which has re-

sulted from the scurvy and concurrent rickets. As the infant improves, the orange juice and beef juice are continued and cod-liver oil and iron are given. The cod-liver oil is especially valuable in those cases where rickets complicates the scurvy.

## CHAPTER XXXI

### DIABETES MELLITUS

In the infant and young child diabetes, as a rule, runs a rapidly fatal course; the younger the child the more rapid and the more fatal is this disease. It rarely occurs in infancy, but is occasionally seen in the young child.

The **etiology** and **pathology** of diabetes in the child are the same as in the adult.

**Symptomatology.**—Among the earliest symptoms noted are thirst, frequent urination, loss of weight and strength, lassitude, and anemia. An increased thirst and appetite causes the child to drink large quantities of water and to eat more than the normal amount of food. An excessive quantity of urine is passed, having a high specific gravity, containing large quantities of glucose, and later acetone, diacetic, and oxybutyric acids are found. As the disease progresses the child loses in weight and strength, its appetite begins to fail, its insatiable thirst continues, the intake of water becomes greater and greater, the quantity of urine is proportionately increased, it becomes irritable and restless, the skin and mucous membranes are dry, furunculosis may develop, an acetone odor may be detected on the breath, and finally a somnolence followed by diabetic coma may terminate in death.

**Treatment.**—The treatment of diabetes in the child is the same as in the adult. The quantity as well as the quality of the food is important. The child should be given only a sufficient number of calories to supply its nutritional needs, and the diet should be made up of the ordinary diabetic foods prescribed for adults. In beginning the treatment it is even more important than it is in the adult that the patient should be placed upon a strictly diabetic diet and that the quantity of water which the child takes in twenty-four hours should be limited as much as possible. If the urine, upon a strict diabetic diet, can be made sugar-free there is a chance that under careful dietetic management life may be prolonged for many years, and that a cure may be effected in a small percentage of cases. It is necessary, however, after the urine has remained sugar-free for a few weeks, that certain carbohydrates should be added for the purpose of developing a tolerance for this class of foods. The carbohydrates which are thus to be added in small quantities to the diet should be first oatmeal, then potatoes, and later small quantities of wheat bread. The dietetic management, however, of these cases cannot be discussed in detail here, since this chapter is a long

one and may be found in any modern textbook on medicine. The point to be emphasized is that unless the child can be got into a condition in which it can metabolize certain carbohydrates such as those mentioned, without causing an increase of sugar in the urine, the case is a hopeless one.

## CHAPTER XXXII

### RECURRENT VOMITING, RECURRENT CORYZA AND MIGRAINE

### RECURRENT VOMITING

**Synonyms.**—Cyclic vomiting, lithemic vomiting, migrainous gastric neurosis, periodical vomiting, bilious vomiting, vomiting with acetonemia.

**Definition.**—Recurrent vomiting, which is one of the most common diseases of early childhood, is an autointoxication produced by systemic and probably intestinal toxins. It is characterized by *recurring attacks* of nausea, persistent vomiting, prostration, and the appearance of acetone bodies in the urine.

**Etiology.**—Liver Incompetency.—The failure on the part of the liver to neutralize or destroy systemic and intestinal toxins is, I believe, the most important cause of this disease. This hypothesis assumes that the liver, from various causes, the chief of which is overwork, more or less suddenly develops a functional incompetency which renders it incapable of converting ammonia and the purin bodies into urea and destroys its so-called filtering function, which normally renders innocuous the fermentation products which pass through it from the intestinal canal. As a result of this liver inactivity both systemic and intestinal toxins escape into the general circulation and produce an autointoxication which is named from its most prominent symptom "Recurrent Vomiting." After a few hours or days, as the case may be, the liver resumes its function and the acute attack of autointoxication is ended. In those very rare cases where this condition terminates fatally there is a well-marked fatty degeneration of liver cells. Howland's and Richards' investigations indicate that the chief metabolic disturbance underlying recurrent vomiting is deficient oxidation and also that the products of intestinal fermentation (indol, etc.) are more or less directly responsible for the symptoms. There are many factors, predisposing and exciting, which in individual cases help to produce the liver incompetency which causes this widely varying symptom group. This variability may be explained by the fact that it is not always produced by the same autotoxins. In one group of cases the intestinal toxins may dominate, and in another the systemic; in still another the autointoxication may be almost or quite overshadowed by nervous symptoms produced by powerful exciting causes. In these cases the symptoms of hysteria and other neuroses may be commingled with those of autointoxication.

Exciting Causes.—*Overeating* is perhaps the most common of all ex-

citing factors. Individual idiosyncrasies with reference to the metabolism of certain food stuffs are most important; defective carbohydrate metabolism is very commonly present in infants and children, to such a degree that an excess or even ordinary quantities of sugars and sometimes of starches is quickly followed by attacks of autointoxication; an inability to metabolize excessive quantities or even normal quantities of fat is also very common; defects in protein metabolism are less rarely seen. In some instances, following a "food injury" to the metabolism from an excessive intake of either fats or sugars, the child for months or years may not be able to take even comparatively small quantities of one or other of these foods without producing an attack of recurrent vomiting. In time, however, under careful feeding it may gradually recover its normal powers of metabolizing these foods. Food idiosyncrasies are not confined to fats and sugars; acid fruits, certain vegetables, eggs, or milk may in individual cases be followed by attacks. The trouble in such cases is not solely a question of digestion, but also one of metabolism. Food idiosyncrasies, therefore, must be looked for in every case.

Among other exciting causes may be mentioned mental and physical fatigue, mental excitement, nervous strain, fright, anger, acute infections, general anesthesia, especially by ether, and severe reflex irritation originating in the eye, nasopharynx, or genitourinary organs.

The acidosis which occurs in recurrent vomiting is, in most cases, a very important part of the pathological process and when well pronounced no doubt contributes to the production of the clinical syndrome of this disease. It may occur early or it may appear late, and in a small minority of cases it is not present at all. It is not to be considered as an etiological factor, but rather as an important symptom of this condition. The pathology of acidosis is elsewhere discussed.

Predisposing Causes.—Heredity is a most important predisposing factor; a family history of migraine, gout, or neurotic disease is present in most cases. Constipation is nearly always present. Recurrent vomiting occurs more frequently among children of the upper classes. Mental overwork and nerve excitement, combined with indoor life and confinement in ill-ventilated rooms, are important factors. The great majority of cases occur during infancy and childhood. I have seen the first symptoms appear as early as the third month, but it is most commonly seen between the third and tenth year; after this period the tendency is to spontaneous recovery, or to a change in form of the autotoxic attacks in which vomiting plays a secondary rôle. They may gradually be transformed in the older child and adult into true migraine. They are slightly more common in girls than in boys and are seen more frequently in winter than in summer.

**Symptomatology.**—General Symptoms.—The following description presents the ordinary type of this disease. There is usually a prodromal period lasting from a few hours to a few days. This may be characterized by sallowness of complexion, dark rings under the eyes, general malaise, constipation, coated tongue, disagreeable odor to the breath, loss of appe-

tite, gastric discomfort, anorexia, nausea, general nervous irritability, sleeplessness, flushing of the cheeks, and possibly coryza, dyspnea, and sighing respirations. The stools may be white or putty-like in color with a disagreeable odor. Not all of these prodromes are present in any one case, but in an individual case the same prodromal symptoms commonly precede the recurring attacks. Occasionally, without warning, the attack may be ushered in with vomiting, quickly followed by fever. In some of my cases the attacks were always preceded by a vasomotor coryza.

*Vomiting.*—This is the most constant and most characteristic symptom. In the beginning it may not be severe, but in a few hours it may become very violent and associated with retching, the vomitus containing *hydrochloric acid,* mucus, bile and rarely blood; in the interval between the attacks there may be nausea. The vomiting may continue for a few hours, or it may last eight or nine days; when it continues for any length of time it produces great emaciation, is associated with great prostration and is accompanied by insatiable thirst. When the vomiting subsides the gastrointestinal canal quickly resumes its functions; food is taken without the slightest discomfort, convalescence is rapid, and, within from four to nine days, the patient has fully recovered. Subsequent attacks occur at irregular intervals, and it is this recurrence which leads to their differentiation from acute gastritis. When second or third attacks of vomiting, in which the same symptom group is reproduced, occur in spite of careful feeding and without apparent cause, the physician must suspect their autotoxic character and make the necessary examinations of the urine and stomach contents which confirm the diagnosis. While it is the rule that patients suffering from recurrent vomiting have little or no gastric or intestinal disturbance in the interval, yet this is a rule which has many exceptions, especially in children under four years of age. In the very young child these autotoxic attacks may manifest themselves by a difficulty in digesting cow's milk, and gastric indigestion associated with vomiting and mild intestinal fermentations may intervene between the more pronounced attacks of recurrent vomiting.

*Pain* is usually absent; this is especially true of recurrent vomiting as it occurs in the child. In those cases where the autotoxic attacks are continued into adult life severe pain in the head or stomach may be associated with the vomiting.

*Constipation,* which commonly precedes the attack, becomes more obstinate as the attack goes on, and it is one of the most difficult symptoms to relieve because the irritable condition of the stomach will not tolerate cathartic medication. When the constipation is relieved by cathartics the discharges are putrid. Diarrhea occurs in rare instances.

In severe cases the *emaciation* and *prostration* are rapid and extreme; the abdomen is boat-like or flattened, the eyes are sunken and the face has an anxious expression.

*Fever* from 101° to 105° F. is present in nearly every case; the younger the child the more marked the febrile reaction. In the older child the tem-

perature may not rise above normal. After the second or third day the temperature subsides and may become subnormal. The *pulse* is rapid and usually irregular. The breathing may be but slightly disturbed, or it may be rapid and panting; in some cases there is dyspnea with wheezy respiratory sounds. The peculiarly sweet and rather offensive *acetone odor* of the breath is a striking symptom; as a rule it occurs early, and in a few instances is never present. When once observed, it is easily recognized and may be so penetrating that it is noticed on entering the room. As the disease progresses the tongue, pharynx and lips become dry and irritated. Toward the close of a severe attack there is a tendency to somnolence, and a prolonged sleep is frequently followed by the first indications of improvement.

Children who suffer from recurrent vomiting are usually precocious and neurotic. They present varying degrees of general nervous excitability and restlessness, even in the interval between the attacks. In very nervous children, as Snow has noted, convulsions may occur.

Urine.—The acetone bodies are the most characteristic findings in the urine. In the milder cases acetone alone may be found, in those of moderate severity both acetone and diacetic acid, and in more severe cases oxybutyric acid is also present. The urine is scanty, concentrated and hyperacid and in severe cases albumin and hyalin casts are present. Indican, indolacetic acid, uric acid, and the xanthin bodies are markedly increased during the attack. Howland and Richards report an increase in the unoxidized sulphur and a diminution in sulphuric acid.

The Blood.—A leukocytosis of 16,000 to 20,000 commonly occurs, with a relative increase in the small lymphocytes.

**Diagnosis.**—The periodical return of the symptom group is a most important diagnostic indication. The presence of free hydrochloric acid in the vomited matter may materially assist in differentiating this condition from acute gastritis, and the urine findings above noted, with the absence of pain and abdominal tenderness, should differentiate it from appendicitis and intestinal obstruction.

**Prognosis.**—It should be remembered that this is one of the most common disorders of childhood and that in most instances it will be overlooked if systematic examinations of the urine for acetone and diacetic acid are not insisted upon. The prognosis, as far as recovery from the attack is concerned, is good; death, however, may occur from exhaustion or from a terminal nephritis; only a few fatal cases have been reported. The prognosis as to the prevention of these attacks is also good. Under proper medical supervision the attacks cease, the improvement in the child's general health continues, and as it grows older its nervous system becomes more stable and a tendency to these recurring attacks is thus outgrown. Untreated cases may later be transformed into migraine or, rarely, into epilepsy.

**Clinical Types.**—It should be understood that there are many variations in the clinical syndromes grouped under the general heading Recurrent

Vomiting. The attack may not proceed beyond the prodromal symptoms; in many cases there may be little or no vomiting, and the characteristic syndrome may be marked by a periodic return of fever lasting one or two days, associated with a coated tongue, bad breath, lack of appetite, nausea and constipation. In other instances, especially in older children, the prodromal symptoms of recurrent vomiting may occur associated with nausea, headache and narcotism, and in still another group the same prodromal symptoms may be associated with a recurrent coryza or a recurrent asthma, which may or may not be accompanied by nausea and occasional vomiting. If one remembers that the symptom group above outlined may present itself in all grades of severity, but that in the same individual these attacks closely resemble one another, there will be little difficulty in making a diagnosis.

There is another syndrome associated with a marked acidosis described by Thomas D. Parke, which my experience leads me to believe is a distinct clinical entity due to some severe toxemia. It occurs most commonly in children under three or four years of age. The symptoms in the beginning are those of acute gastrointestinal infection. There is diarrhea and nausea and commonly an acetone odor to the breath. The stools usually contain mucus and blood, are passed with more or less straining and are frequently preceded by intestinal colic. Labored and rapid breathing is a prominent symptom. The liver is enlarged. There may be a slight fever, but as the disease progresses the temperature becomes subnormal. There is marked prostration and the disease commonly comes to a fatal termination with an increasing gastrointestinal irritation and a marked increase of the acetone bodies in the urine. This symptom group is much more severe, much more dangerous and differs materially from the ordinary syndrome of recurrent vomiting. The fatal cases terminate within three or four days after the onset of severe symptoms. The postmortem findings show an enlarged liver, which has undergone fatty degeneration, and there may be fatty degeneration of other organs. Apart from this the pathological findings are not definite.

**Treatment.**—Treatment of the Attack.—If seen in the prodromal stage, one-fourth of a grain of calomel and five grains of bicarbonate of soda should be given every half hour until two grains of calomel are taken, and two or three hours later a saline laxative should be given. This should be followed by five or ten grains of bicarbonate of soda every two or three hours over a period of several days, administered in carbonated water, plain water or peppermint water. No food whatever should be allowed for at least twenty-four or thirty-six hours, or until the nausea and vomiting have been controlled. After the attack is well on the nausea and vomiting may preclude not only all food, but all stomach medication. The calomel and bicarbonate of soda, however, may be tried at any stage of the attack, and if the nausea and vomiting are not aggravated they may be continued. At intervals throughout the attack water may be allowed in small quantities, even though the stomach rejects it; when the patient

is able to retain water, then small quantities of thin beef broth may be given. If water is not retained by the stomach it is advisable to give, at intervals of six or eight hours, high rectal enemata of 6 or 8 ounces of physiological salt solution, or of a 1 per cent. bicarbonate of soda solution. Edsal's suggestion that large doses of bicarbonate of soda be given by the mouth is a good one in those cases where the soda is retained, but the great discomfort and exhaustion which follow attacks of vomiting teach us that it is wise, when the stomach is very irritable, to let it have a period of prolonged rest and then attempt to give bicarbonate of soda by the mouth in 8- or 10-grain doses every two or three hours. In the most aggravated cases, where prostration is extreme and vomiting has continued over a number of days, 8 to 16 ounces of sterile physiological salt solution combined with five or ten grains of bicarbonate of soda to the ounce may be injected into the subcutaneous tissues. In this same type of case the hypodermic use of morphin frequently controls the vomiting, and may, like hypodermoclysis, be a life-saving measure. Small doses of from 1/20 to 1/60 of a grain of morphin, depending upon the age of the child, are usually sufficient to control the irritability of the stomach long enough to allow the bicarbonate of soda solution given by the mouth to be absorbed. When necessary the morphin and the hypodermoclysis may be repeated at intervals of eight to twelve hours.

Interval Treatment.—When the child is convalescent causes of reflex irritation to the nervous system should be carefully sought for and removed. Constipation, which is usually present, must be relieved; this may be done by palatable solutions of sulphate and phosphate of soda. These saline laxatives are advisable in beginning the treatment; later cascara sagrada, rhubarb and other cathartics may be used; enemata are not to be relied upon. Abdominal massage may relieve the constipation. General massage is one of our most valued remedies in overcoming the constitutional conditions which predispose to recurrent vomiting; it is especially indicated in patients of feeble constitution who are not strong enough to enjoy the benefits of outdoor life and active exercise.

In the early interval treatment of this condition the wintergreen salicylate of soda and the benzoate or bicarbonate of soda put up in palatable solution are our most valued remedies; two grains of the salicylate and five grains of the bicarbonate may be given to a child six years of age over a period of months. After three or four months these remedies may be given once or twice a day for a year or more, as the indications may direct. If during this time the prodromal symptoms of an attack make their appearance, the calomel and bicarbonate of soda are to be given as previously directed, and then the salicylate and bicarbonate of soda are to be resumed. From a very large experience I have the greatest faith in the efficacy of the interval-medical treatment as here outlined. It may be necessary occasionally to interrupt the alkaline treatment and substitute such tonics as malt and arsenic; after a time, however, it is necessary to return to the alkaline treatment.

In the treatment of recurrent vomiting in older children I use the formula which I originated many years ago for the treatment of migraine. It is as follows:

| | |
|---|---|
| Sodii sulphatis (dry) | 30 grains |
| Sodii salicylatis (from wintergreen) | 10 grains |
| Magnesii sulphatis | 50 grains |
| Lithii benzoatis | 5 grains |
| Tincturæ nucis vomicæ | 3 drops |
| Aquæ destil. to make | 4 ounces |

This prescription is put up in siphons and charged with carbonic acid, and the child is directed to take, half an hour before breakfast, a sufficient quantity to produce at least one bowel movement during the morning. This prescription is a remedy of great value in the preventive treatment, not only of recurrent vomiting, but also of migraine; it may in fact replace all other medication.

Dietetic Treatment.—This is of very great importance. In beginning the treatment all sweets, fats, raw fruits, strawberries, rhubarb, tomatoes, salads, tea, coffee, beef-juice, beef-tea, pastry, gravies, cream, cod-liver oil and alcohol are to be avoided, and the child should not be allowed to eat large quantities of meat. The following foods may be recommended: skim-milk, vegetable soups, cereals, well-cooked vegetables, cooked fruits, bread, eggs, fish, chicken, mutton and beef. It is most important that children suffering from recurrent vomiting should be guarded against an *excess of food* of any kind and that *sweets of all kinds* should be carefully excluded. It will be found that in certain instances the sweets are the prime cause of the trouble, while again in other cases the attacks cannot be controlled until the fats are eliminated from the diet. The diet of the child should be carefully balanced; if, for example, he happens to be an excessive meat eater, the meats should be somewhat restricted and a proper proportion of vegetables, cooked fruits, or cereals given, and the child should also be made to cultivate the habit of drinking as much water as possible.

Hygienic and Climatic Treatment.—As suboxidation is one of the essential underlying pathological processes of this disease, it is necessary that the child should have as much fresh air and outdoor exercise as possible. Most of these children prefer an indoor life and intellectual pursuits, so that it becomes necessary for the physician to give special directions with reference to *open air sleeping apartments* and the number of hours of outdoor play which the strength of the child and the season of the year will permit. A change of climate in many instances may be advisable to avoid the extreme heat of summer and the damp cold of winter. These children should, as a rule, be taken out of school. Mental stimulation, nervous excitement and all forms of mental and physical fatigue should be avoided until their physical and nervous condition justifies a return to the ordinary routine of child-life.

## RECURRENT CORYZA

There is a form of coryza, recurring at irregular intervals without apparent local or external cause, which is self-limited and is closely related in its etiology and pathology to recurrent vomiting.

**Symptomatology.**—Constipation, loss of appetite, general nervous irritability and sallowness of skin may be prodromes. The attack itself comes on with an acute congestion of the nasal mucous membrane, accompanied by a profuse, irritating, thin mucous discharge from the nose, which produces redness and swelling of the lip over which it flows; at the same time there is commonly an acute congestion of the mucous membranes of the eyes, marked by a redness and swelling of the conjunctiva, intense photophobia, and a profuse overflow of tears. These symptoms come on rapidly and are associated with a state of extreme nervous irritability. The patient seeks a darkened room, buries her head in the pillows, or shields her eyes with her hands when light is admitted. These attacks are self-limited; the symptoms continue in the worst cases for four or five days, and then quickly subside. Convalescence is very rapid; within two or three days after the symptoms begin to disappear the patient is quite well, showing little or no evidence of disease of the mucous membranes, which were so recently the site of extreme irritation. These attacks recur from time to time at irregular intervals, very like those of recurrent vomiting and migraine, and in the interval between the attacks there may be no evidence of disease of the mucous membranes of the eye and nose. The above description represents the severe type of this disorder. In milder cases the attack may manifest itself as a more or less severe coryza without the eye symptoms, and may in this form occur as one of the prodromes of an attack of recurrent vomiting. Vasomotor coryza is not uncommonly associated in its clinical manifestations with an urticaria of the skin.

The **treatment** in every particular is similar to that of recurrent vomiting.

## MIGRAINE

Migraine is an autointoxication due to systemic or intestinal toxins which find expression in recurrent self-limited attacks of severe paroxysmal headaches, usually unilateral, commonly accompanied by nausea, vomiting, vertigo, and visual phenomena and followed by a profound sleep from which the patient awakes free from pain.

Migraine is not often a disease of early childhood. The great majority of cases appear in late childhood or early adult life.

It is very similar in its etiology and pathology to recurrent vomiting. It may, however, be noted that reflex factors such as eye-strain, diseases of the nasopharynx and of the genitourinary and pelvic organs, play a more important rôle as exciting causes in touching off an attack of mi-

graine than they do in producing recurrent vomiting. I have in a number of instances seen the recurrent vomiting attacks of early childhood become attacks of true migraine in late childhood and adult life.

The treatment of migraine occurring in childhood is similar to that above outlined for recurrent vomiting.

# SECTION VI

## *INFECTIOUS DISEASES*

## CHAPTER XXXIII

### FEVER

Fever is the most common symptom of illness in infancy and childhood. At this period of life there is such a predisposition to fever that high temperatures may occur from comparatively slight causes, and for this reason the direct exciting cause of the fever is not always apparent at the beginning of the child's illness. The physician must frequently wait until the second day, or perhaps later, before he can determine the nature of the pathological disturbance. In the meantime he must prescribe for the sick child and be directed in the selection of a diet and other remedies along such lines as his general experience teaches him are the safest and the most likely to bring good results under existing conditions. In thus prescribing for a syndrome of which fever is the most important symptom, the physician's judgment must be largely directed by his knowledge of the most common exciting causes of fever at different ages in the life of the child, as well as by the accompanying but as yet inconclusive symptoms with which the fever is associated. For the above reasons an inquiry into the most common direct causes of fever at different periods in the life of the child should be of the greatest practical importance to physicians in enabling them to begin the treatment of these cases in a way to give the most satisfactory results.

In the chapter on Growth and Development I have discussed the physiological peculiarities of the heat-regulating mechanism of the young nervous system and have there shown that the tendency to high fever from comparatively trivial causes at this time of life is due to the marked excitability of the thermogenic centers and the feeble control which the inhibitory centers exercise over them, and have also called attention to the fact that the very efficient heat-dissipating mechanism of this period of life acts as a protecting agency, and by its quick response reduces these high temperatures. This rapid play of function between the heat-generating and heat-dissipating functions accounts for the great variability of the temperature curve which characterizes the fevers of childhood. A sustained temperature with little variations is rarely seen in the infant or

child, except in lobar pneumonia and typhoid fever, and even in these we have greater variations in the temperature curve than we do in the same conditions in the adult. In the same chapter I emphasized the fact that the tendency of the individual child to high and variable temperatures might be greatly exaggerated by a neurotic inheritance, a chronic malnutrition or unfavorable environment. It is evident, therefore, that apart from the unfinished and unstable condition of the heat-regulating mechanism of the child, the most important predisposing causes of fever are to be found in all those conditions which produce the malnourished and abnormally nervous child. These have been previously discussed in the chapter on The General Hygiene of Infancy and Childhood. This leaves for our discussion here the direct exciting causes of fever.

**Exciting Causes of Fever.**—The most common exciting causes of fever in infancy and childhood may be classified as follows:

1. Intestinal toxemia, commonly of bacterial origin, but including also the "food injuries" described under Acute Intestinal Indigestion.
2. Systemic toxemia of bacterial origin.
3. Systemic autointoxication of non-bacterial origin.
4. Heat-stroke.
5. Mechanical and reflex irritation, including simple indigestion.
6. Muscular action (convulsive) and over-fatigue.

Intestinal toxemia is by far the most common and the most important cause of fever in children *under two years of age;* this is especially true of artificially fed infants. This fact alone is of the very greatest importance in directing the physician along proper lines of therapeutic action. In a child under two years of age, especially if it is being fed upon artificial food, elevation of temperature, the causes of which cannot be ascertained, may be assumed to be due to intestinal toxemia and treated accordingly until a positive diagnosis can be made. There are many other causes which may produce fever in children at this time of life, but the fact remains that under the above-named conditions a tentative diagnosis of gastrointestinal toxemia is shown by later developments, in the great majority of cases, to be the true one, and above all it offers a safe and wise course for therapeutic action. The influence of free catharsis and abstinence from food upon the temperature curve in these cases will materially assist in confirming the diagnosis. If under these measures the temperature falls and remains low, it is a safe inference that intestinal intoxication was the cause of the fever, but, if following this treatment there is no fall in the temperature, or if following the fall there is a subsequent rise of the temperature, which is not influenced by catharsis and starvation, the inference is that the fever with its accompanying symptom group is due to other causes than intestinal intoxication.

Systemic intoxication of bacterial origin is the most common cause of fever in children over two years of age, and the older the child the more important becomes this factor as a fever producer. In children over three years of age, an elevation of temperature without apparent cause

commonly means the child is suffering from tonsillitis, influenza (la grippe), pneumonia or one of the other acute infectious diseases. Whatever may be the subsequent course of the temperature curve in the various acute infections, they are almost always announced by an early rise in temperature, and the distinctive symptoms which complete the symptom group and make possible an accurate diagnosis may not appear until the second or third day, or even later. In these cases it is always wise to isolate the sick child from the well ones in the family. A preliminary cathartic, a light diet, and some such medicine as aspirin or phenacetin, to control the fever and nervous symptoms, are indicated until the diagnosis is made.

Systemic autointoxication of non-bacterial origin plays a rather important rôle in producing fever in infants and young children. The fever, however, from this cause cannot be differentiated from that produced by one of the acute infections until symptoms, such as occur in uremia, acidosis and other intoxications, present themselves to complete the autotoxic syndrome. The clinical picture produced by these conditions is elsewhere described under Recurrent Vomiting. The preliminary treatment of these cases, even if we knew from the beginning the character of the disease, would be that of free catharsis and abstinence from food.

Heat-stroke, as Forchheimer has long taught, is an important cause of fever in infancy and childhood. Probably the best explanation of the fever of heat-stroke is that the feeble inhibitory heat centers of the child are still further weakened by the heat, so that practically no restraint is exercised over the thermogenic centers. This explains the fact that the younger the infant the more prone is it to have elevations of temperature from exposure to excessive heat. In the premature infant the body temperature may be raised far above normal by the unwise application of hot water bottles and other forms of external heat. During the summer season many of the so-called cases of cholera infantum with high temperatures and other severe symptoms are due to excessive heat. In these cases the heat acts directly as a fever producer and also indirectly in keeping up the fever by producing a gastrointestinal fermentation. High fever in a young infant that has been exposed to unusual heat should be attributed to this cause, and should be treated by ice-bags to the head, tub-baths, free catharsis and abstinence from food until the character and cause of the fever have been definitely determined.

Direct mechanical and reflex irritations may produce an elevation of temperature in the young infant. It is important to remember that a purely reflex fever may occur during infancy. These fevers are, as a rule, evanescent and of comparatively little pathological importance. They occur very infrequently in normal, well-nourished infants, but they are of very common occurrence in nervous, malnourished ones. The cutting of a tooth, undigested food, worms, foreign bodies in the intestinal canal, and excessive pain (earache) are among the most common causes of reflex elevations of temperature. Slight elevations of temperature

therefore occurring in nervous, malnourished infants may be due to the coming through of a tooth, or to undigested food in the intestinal canal, and elevations of temperature with violent and prolonged paroxysms of crying should lead to a careful examination of the ear. The preliminary treatment, however, of these cases is the same as that previously outlined for intestinal toxemia, and their subsequent history will establish the diagnosis and direct more specific treatment.

Excessive muscular action may cause an elevation of temperature in infants and young children. The manifestation of muscular energy is always accompanied by the formation of heat, and excessive muscular action, such as occurs in general convulsions, may be accompanied by an increase in the body temperature; the elevation of temperature, therefore, at the close of a convulsion may be higher than at its beginning. This cause of increased temperature, however, is of comparatively little importance. Over-fatigue may cause an elevation of temperature in nervous, malnourished children. The rise in temperature from this cause usually occurs in the afternoon and may reach to 101°F. A slight afternoon rise of temperature in this type of child should be treated by fresh air and rest for a few days, and if the fever still continues it is due to other causes.

In the above study of the causes of fever I have attempted to furnish the data which will assist one in arriving at a fairly accurate determination of the cause of a fever occurring as the initial symptom of an acute illness, and I have also attempted to indicate the safe and rational therapeutic measures with which the treatment of acute febrile cases should be begun. There is in the obscure fevers of infancy and early childhood a field of diagnostic and therapeutic inquiry quite as important as that upon which we have just dwelt. In the "obscure fevers" the difficulty in diagnosis continues after the onset of the initial symptoms. In some of these cases the failure to make the diagnosis is due to carelessness, insufficient knowledge, or lack of facilities on the part of the physician; in others the symptoms and physical findings do not form a syndrome sufficiently clear to warrant a definite diagnosis. For these reasons it is important that the physician should have constantly in mind the most common causes of, and the most rational treatment for, these "obscure fevers."

**Obscure Fevers of Infancy and Childhood.**—Holt's (inanition) fever is the most common cause of fever during the first four or five days of life; in fact, fever rarely occurs at this time from any other cause. Holt's fever is a clearly defined syndrome; the elevation of temperature usually occurs on the second or third day of life and disappears by the fifth or sixth; it requires no treatment other than the giving of water or breast-milk. It is elsewhere described.

Sepsis is the most important cause of fever during the second week of life. Continuous fever occurring at this time, not associated with digestive disturbances, is in the great majority of instances due to sepsis, and the septic infection commonly finds an entrance through the open um-

bilical wound. Since sepsis in the new-born is a disease of great gravity, an unexplained fever during the second week of life is sufficient cause for alarm and demands that other symptoms of sepsis should be carefully looked for and that the treatment for this disease should be at once instituted.

Lobar pneumonia is a common cause of obscure fever in the infant. The fever of this disease is not only high, but is more sustained than that of any other fever of infancy. A high temperature curve that runs its course with little variation, occurring in an infant under two years of age, is strongly suspicious of lobar pneumonia. This fact is important, since in many cases of lobar pneumonia in the infant the physical signs are not discoverable until the fourth or fifth day. A sustained high temperature, therefore, occurring without apparent cause in an infant, should be treated as a lobar pneumonia until a definite diagnosis is made.

Otitis media is perhaps the most common cause of obscure fever in children under two years of age, and is not an uncommon cause in older children. The fever of otitis media is subject to wide variations; at one time during the day it may reach 104° or 105°F., and at another be almost or quite normal. Otitis media as the cause of fever is perhaps more generally overlooked than any other disease; for this reason it should be a rule of practice to examine the ear and look for other signs of otitis media in all the unexplained fevers of infancy and early childhood. It is not always associated with earache, but when this symptom is present, the violent fits of crying should at once call attention to the ear as the cause of trouble.

Pyelocystitis, although nothing like so common as pneumonia or otitis media, is not an uncommon cause of obscure fever in infancy and early childhood. Unless the physician be on the alert, these cases are nearly always overlooked and the fever is attributed to some other cause. The fever of pyelocystitis is much more irregular than that of lobar pneumonia and very commonly there are few or no symptoms to call attention to the genitourinary organs. For this reason, unless the physician makes it a rule to examine the urine in all the obscure fevers of infancy and early childhood, these cases may run on for weeks without attention being called to the bladder or kidneys as the site of the infection.

Tuberculosis is the most common cause of continued fever of obscure origin in children over four years of age. The tuberculosis of childhood, as I have elsewhere said, is usually concealed, and one of its earliest manifestations is a slight irregular fever. As the disease progresses the temperature may rise to 103° or 104°F., but it commonly falls to normal or below normal during the day. The tuberculosis of infancy (that is to say under two years of age) is not a concealed disease; it runs a rapid and much more serious course and is not commonly manifested as an obscure fever. In children, therefore, over four years of age a long-continued irregular fever, without apparent cause, should strongly suggest tuberculosis, and the diagnosis can usually be confirmed by other signs and symptoms outlined in the chapter on that disease.

Typhoid fever is not an uncommon disease after the second or third year of life. During childhood it may be classed among the obscure fevers, since the nervous and abdominal symptoms, which are so characteristic in the adult, are commonly absent in the young child. The fact that typhoid fever in the child, like lobar pneumonia in the infant, is the most common cause of high and sustained fever is very important from a diagnostic standpoint. Every fever occurring at this period of life that runs a high course with comparatively little variation should be tentatively diagnosed as typhoid and treated as such until further developments make the diagnosis clear. The Widal reaction commonly makes the diagnosis in these cases, but even in the presence of a negative Widal the case should be treated as typhoid until other causes for the fever are discovered. In some instances the Widal test is not positive until the second or third week of the disease, and then again there is a group of cases produced by the paratyphoid, the bacillus enteritidis, and perhaps other organisms, which have all the clinical characteristics of typhoid fever and yet never show the Widal reaction.

Subacute and chronic intestinal toxemia may be the cause of obscure fever in infancy and early childhood. In these cases a temperature from 101° to 101½°F. may be present in the afternoon with a normal or subnormal temperature in the morning, and there may be little, on casual examination, to call attention to the intestinal canal as the cause of the disturbance. On closer inspection, however, it will be found that the intestinal discharges are not normal. They may be fragmentary, putrid, and covered with more or less mucus, and an examination of the urine will show a marked increase in indican or indolacetic acid. Suitable cathartics from time to time with a carefully regulated diet will control the temperature if intestinal toxemia is the exciting cause.

Septic infection is not an uncommon cause of obscure fever throughout childhood. The temperature curve of sepsis, which runs from 103° to 105° F. at one time during the day and falls to 96° or 97° F. at another, is so suggestive of sepsis that septic infection is commonly suspected with this type of temperature, although the localization of the sepsis may be very obscure. In these cases the presence of an increasing polynuclear leukocytosis may confirm the diagnosis. If an examination of the ear excludes otitis media, the bronchial or cervical lymphatics or the mastoid may be suspected as the possible site of the sepsis. In older children the antrum or frontal sinus may harbor the infection, and in some cases we may have a general septicemia which runs its course to a fatal or favorable termination without apparent localization. In the great majority of instances septic infection in infancy and childhood occurs as the sequel or as a complication of one of the acute infectious diseases; under such conditions the physician, being prepared for the development of septic symptoms, rarely fails to make the diagnosis, although he may have trouble in locating the focus of infection.

Treatment.—In the treatment of obscure fevers it is wise to begin

with a cathartic such as small doses of calomel followed by castor oil. This preliminary clearing of the intestinal canal can do no harm in any form of fever and is of value in reducing the temperature even though the intestinal canal be not the site of the disease. Throughout the course of the fever the intestinal canal should receive careful attention; constipation, intestinal fermentation and diarrhea are to be treated by appropriate remedies, whatever may be the cause of the fever.

*Diet.*—A fluid diet, free from milk or other albuminous foods, should be prescribed for the first twenty-four or thirty-six hours, or until it has been determined that intestinal intoxication is not the cause of the fever. In children over three years of age having a sustained temperature, a typhoid diet should be prescribed until it is proven that the disease is not typhoid; this rule will prevent many gross dietetic errors. Jacobi says: "In ordinary fevers the food must be liquid and rather cool; in vomiting, cold; in respiratory diseases, warm; in collapse, hot. The best feeding time is the remission; in intermittent fevers nothing must be given during the attack except water, or acidulated water, now and then with an alcoholic stimulant; in septic fevers, nothing during a chill, except either cold or hot water, according to the wishes of the patient, with alcoholic stimulant. Common ephemeral catarrhal fevers may do without food (except water) for a reasonable time. Sleep must not be disturbed, except in conditions of sepsis and depressed brain action. In both there is no sound sleep, but sopor, which should be interrupted. In sepsis this rousing from sopor is an absolute necessity. Unless they are aroused frequently to be fed sufficiently and stimulated freely, the patients will die. Besides, in most of the cases the temperatures are not high, and there is no contraindication to feeding on that account. Chronic inflammatory fevers bear and require feeding as generous as it must be careful."

*Antipyretics.*—The ice-bag not too closely applied to the head is one of the most valuable measures we have for the control of high temperatures in the infant and child. In the application of this remedy, however, it is necessary that the patient should be under the observation of a competent nurse, since there is some danger in very young and delicate infants that the prolonged application of ice to the head may produce a subnormal temperature. This should be guarded against by careful temperature records, so that when the temperature approaches normal the ice-bag may be removed; in older children this danger does not exist. The ice-bag when properly applied is not only a satisfactory antipyretic measure, but it exercises a very pronounced influence over the nervous symptoms which accompany the fever. The bath is the safest and most effective agency we have for reducing the body temperature. It is important to remember that the cold bath does not always act as kindly in infants as it does in older children. Frail and nervous infants do not react well from the cold bath; the shock to the nervous system produced by the sudden application of cold water to the body may do more harm than good. The temperature of the bath must be regulated by the age and

strength of the infant. In young and delicate infants a warm or tepid bath, or a sponge bath with alcohol and warm water, will, when combined with the use of the ice-bag in the interval between the baths, reduce the body temperature and exercise a sedative influence on the nervous system. In older and sturdier children cool baths and cold packs may be given with signal advantage, but it is rarely necessary to use a bath below 80°F. Phenacetin and aspirin in doses suited to the age of the child may be safely used for the control of the temperature in the early stages of the ephemeral fevers of childhood. These medical antipyretics should be used only during the first twelve or twenty-four hours of the fever and then only for controlling unusually high temperatures which are associated with marked nervous symptoms. They should not be used in the prolonged fevers associated with marked prostration. Fever patients should, if possible, be put to bed and kept there until the temperature has reached normal. They should also be isolated from other children and shielded from all unnecessary excitement.

## CHAPTER XXXIV

### TYPHOID FEVER

Typhoid fever is an acute infectious disease caused by the typhoid bacillus. It is a general infection characterized by a more or less typical temperature curve, and by the involvement of lymphoid tissues, especially Peyer's patches and the spleen.

**Etiology.**—The typhoid bacillus, which is the specific cause of this disease, was first described by Eberth. It is cylindrical with rounded ends, crowned with cylia, and is from 1 to 3 $\mu$ in length and from .5 to .8 $\mu$ in diameter. It has marked motility in liquid media and grows readily at body temperature in common culture media. It is destroyed at 140°F., but resists low temperatures; it can live for months in ice and in ordinary drinking water. On clothing soiled with typhoid feces or urine the bacilli may, if not exposed to light and desiccating processes, live for weeks. This extraordinary vitality under adverse circumstances enables it to live and thrive in every part of the globe from the tropics to the Arctic regions; it is therefore practically a world-wide disease. It is but slightly pathogenic for other animals than man; Grunbaum has produced the disease in chimpanzees. Other living creatures may harbor the bacilli in their intestinal canals and elsewhere, and act as typhoid carriers, even though they present no symptoms of typhoid fever. The typhoid bacillus is to be distinguished from a group of similar organisms to which it belongs, such as the bacillus enteritidis, the colon, and the paratyphoid bacillus A and B, all of which are capable of producing clinical syndromes closely simulating typhoid fever. The bacillus enteritidis and the paratyphoid B are believed to be the chief causes of meat-poisoning.

**Source of Infection.**—It is a generally admitted fact that the intestinal canal and lymphoid tissues of man furnish the most favorable culture conditions for the growth of the typhoid bacilli. The tendency is to an increase in virulence as the bacilli pass from host to host in the course of an epidemic, so that the early cases may be mild and the later ones severe.

**Method of Infection.**—Drinking water, contaminated by the fecal or other discharges from typhoid patients, is the common cause of the spread of this disease. Wells, or the general water supply of cities, may be contaminated by sewage and thus cause epidemics of typhoid fever. Too much stress cannot be laid upon the importance of contaminated drinking water as the great cause of typhoid fever. Milk, being a good culture media, may be a carrier of this infection; many small epidemics have been traced to this source. In such instances the milk is usually contaminated by an infected water supply, the water being used in diluting the milk or washing the pails. Ice-cream made from contaminated milk may produce typhoid fever.

Flies may act as carriers of typhoid infection to milk and other food materials of man. The danger from the fly is greatest in country districts and where large bodies of people are camping under conditions which permit it to come in contact with fecal discharges; in the city, where sanitary plumbing prevails, it has fewer opportunities to act as a carrier. Shellfish, especially oysters and clams which are eaten raw, may be a source of infection when contaminated by sewer discharges. The contagion is rarely, if ever, carried through the air; the surroundings of the patient, except as they are fouled by excretory discharges, are not a source of danger; direct infection, however, does occur to nurses and others who are in close contact with the patient, but such infection is avoidable if proper methods of cleanliness and disinfection are used in the care of the patient. The contaminated bed-clothing, if not properly disinfected, may be a source of danger to the laundress or to the household into which it is carried. Well individuals who have had typhoid fever may, in rare instances, harbor in their intestinal canals typhoid bacilli and thus act as *typhoid carriers,* unconsciously spreading contagion among those with whom they come in close contact.

**Occurrence.**—Typhoid fever occurs most commonly during the months of August, September and October; Osler calls it an autumnal fever. During the first year of life it is comparatively rare, and in the second year it still remains a rather uncommon disease, but thereafter susceptibility rapidly increases up to the twentieth year.

**Pathology.**—Typhoid fever is a general infection; the bacilli of this disease may be found in every part of the body. They are present in the intestinal canal, the blood, the rose-spots, all the viscera and especially in the spleen, mesenteric glands and gall bladder. The lesions produced, while similar to those found in the adult, are much less pronounced and extensive. The solitary follicles and Peyer's patches are enlarged and may be ulcerated. The spleen is greatly enlarged, in nearly every instance

being two or three times its normal size. Slight parenchymatous degeneration of the liver and kidneys, myocardial weakness, hyperemia and even inflammation of the meninges, pneumonia, middle ear suppuration and parotid abscess may occur. As a rule the older the child the more nearly does the pathological anatomy conform to that of the adult type, and the younger the child the more important is the typhoid septicemia.

**Fetal Typhoid.**—The bacilli of typhoid fever may pass from the mother through the placental circulation to the fetus; in about one-half of these cases abortion or premature labor results, and the fetus is born dead; in other cases the child is born with typhoid fever of the septicemic type, and death commonly results within the first week; fetal typhoid is a disease with very great mortality; few cases recover. On the other hand, newly-born infants, born of mothers who have had typhoid fever during the period of their gestation, may show the Widal reaction; in such cases it is probable that the child had typhoid fever *in utero* or that the agglutinating principle in its blood was received through the placental circulation.

**Period of Incubation.**—The Spanish-American Commission found that the average period of incubation for typhoid fever was ten and a half days. In a study of a local epidemic which I reported in 1901, nine days was the shortest and nineteen days the longest period of incubation; the majority had an incubation period of less than ten days.

**Symptomatology.**—General Symptoms.—While in a general way the symptoms of typhoid fever in the child resemble those in the adult, yet it should be remembered that the younger the child the more are these symptoms modified in their course and in their severity, so that the symptom-complex is radically different from that of the adult. In the very young infant there is a general typhoid septicemia without marked local symptoms, but as the child grows older the clinical syndrome gradually changes, giving more and more prominence to the characteristic symptoms as they occur in the adult. It may also be said that the younger the child the more irregular the onset of this disease; in infancy it may be marked by acute prostration, vomiting, sudden rise of temperature and all the evidences of a sudden and general toxemia. In such instances typhoid fever may not be suspected until the symptoms of the acute toxemia have subsided, leaving a continuous fever with other symptoms which suggest the possibility of this disease. In children three or four years of age the onset is commonly marked by headache, a general infection and a gradual rise of temperature. The severity of the disease cannot be predicted from the suddenness or violence of the onset. The course of typhoid fever in young children, if not prolonged by complications, is mild and brief as compared with that in the adult; under three years of age it may not extend over fourteen days; in 80 cases observed by Henoch, 11 lasted less than ten days, 26 less than fifteen days, 16 less than twenty days, 21 from twenty to thirty days, and 6 over thirty days.

*Fever.*—The temperature curve in childhood is not so regular and characteristic as it is in the adult. The first stage may be short, the tem-

perature rising rapidly to its maximum within two or three days, and the third stage or the stage of decline may be marked by great variations, the temperature assuming a markedly remittent or even intermittent type, while the second stage may show an almost continuous temperature with little variation between the morning and evening temperature (Morse). In very young children this sudden rise of temperature with the onset of the disease and its rapid fall toward the close may not be a matter of prognostic importance, but in older children a sudden fall of temperature occurring during the height of the disease should suggest perforation or intestinal hemorrhage. Secondary rises in the temperature after the disease has apparently run its course may result from relapse or from some com-

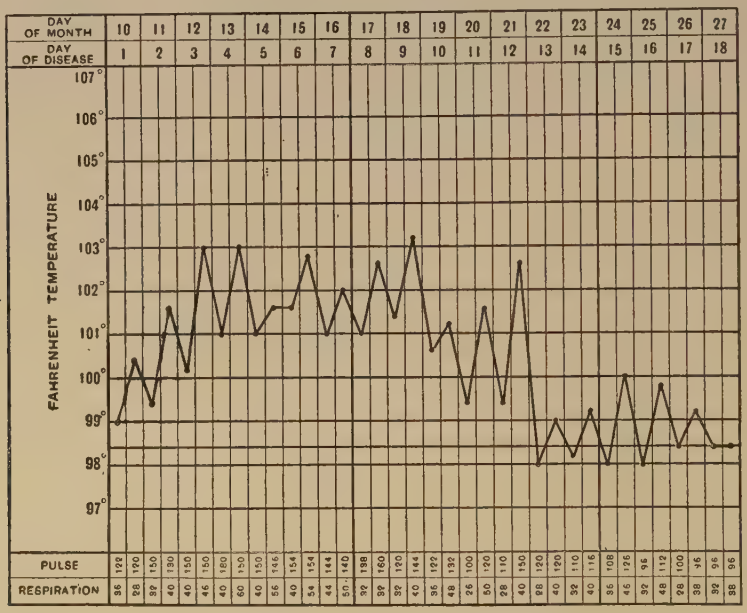



FIG. 42.—TYPHOID FEVER IN CHILD TWO AND ONE-HALF YEARS OF AGE.

plication. Very mild cases of typhoid fever running a short and almost afebrile course are more frequently observed in children than in adults.

The *pulse,* which in the adult is so valuable a prognostic sign, is not of such great importance in the young child. A dicrotic, rapid and intermittent pulse occurring during the height of the disease, in the adult, is a danger signal of great importance. In very young children a pulse of 170 may occur and not be of serious import. In older children the pulse is commonly slow, varying from 70 to 90, but even at this age dicrotism and intermittency do not, as a rule, presage danger. On the whole, therefore, much less information is obtained from a study of the pulse in children than in adults. Systolic murmurs at the apex and rarely at the base are common; they usually appear in the third week of the disease as the acute

symptoms subside, and many continue for weeks after convalescence has been established.

The *exanthem* consists of slightly elevated rose-spots about the size of a pin's head, which disappear on pressure. These spots are distributed over the abdomen and back; in some cases being few in number and widely scattered, in others more numerous and grouped in patches. They commonly appear from the fifth to the eighth day, but may occur earlier or later. In rare instances they may assume a dark blue or hemorrhagic appearance. In some instances an erythema may precede this typical exanthem, but is of no special importance, except that it may complicate the diagnosis. Sudamina may be present; toward the close of the disease fine

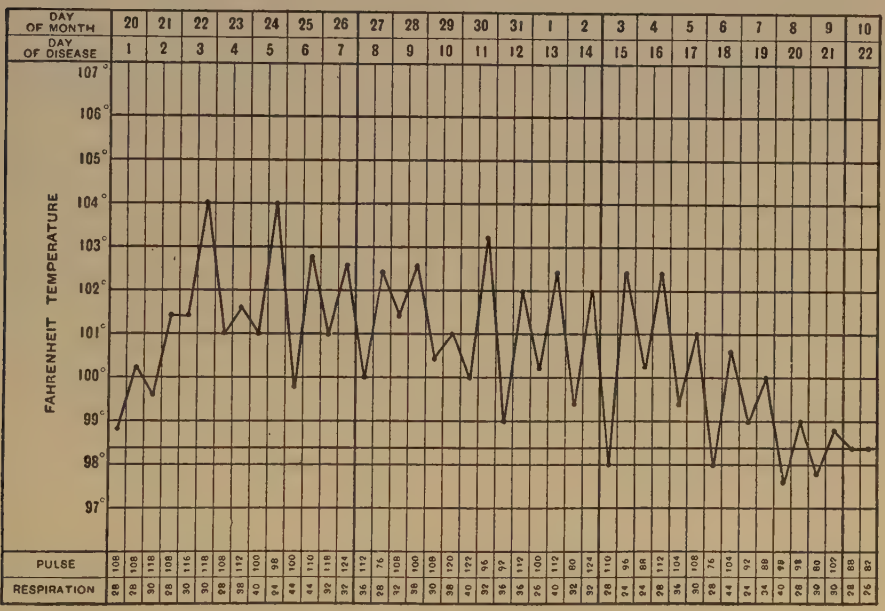



FIG. 43.—TYPHOID FEVER; CHILD SIX YEARS OF AGE.

desquamation occurs. In protracted cases furunculosis, or small subcutaneous abscesses, may be widely distributed over the body and greatly add to the discomfort of the patient, prolonging the disease.

*Enlargement of the spleen* is almost always present in typhoid fever; it begins early in the disease and gradually increases to three or four times its natural size. It can commonly be made out by palpation on the third or fourth day, and for this reason is of very great diagnostic value. It gradually subsides with the other acute symptoms, but it can very commonly be demonstrated after convalescence has been established; in relapses it again increases in size.

DIGESTIVE TRACT.—*Abdominal Symptoms.*—Vomiting occurs much more frequently in children than in adults and is one of the early symp-

toms. Constipation is common, especially between the ages of three and seven, and is usually obstinate enough to require laxative medicines for its relief. It is very frequently associated with gastrointestinal toxemia, which aggravates and prolongs the disease. In quite young children typhoid fever may begin with a sharp diarrhea resembling an acute intestinal toxemia; in these cases, however, the intestinal irritation may quickly subside and the subsequent course of the disease be marked by a mild diarrhea or even by constipation. In older children diarrhea is the rule, the discharges being frequent and having the characteristic pea-soup appearance. Meteorism is rarely a marked symptom in the young child; in older children it may render difficult the palpation of the spleen and the percussion of liver dullness. In the young child abdominal pain

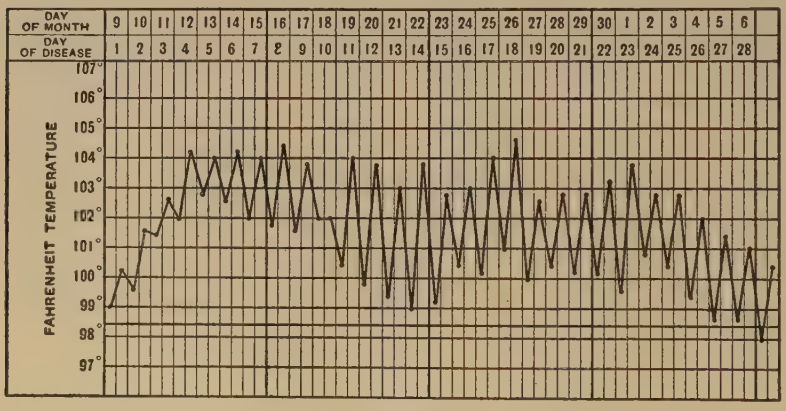



Fig. 44.—Typhoid Fever; Child Ten Years of Age.

is not usually present, and gurgling and tenderness in the right iliac region cannot commonly be made out.

*Intestinal hemorrhage and intestinal perforation* are very rare under six years of age. From the tenth to the fifteenth year, however, they are not so uncommon and are marked by the same symptoms as in the adult.

The *tongue* is white, but the tip and edges are clean and dark red in color; later the coating gradually disappears from before backward, giving it a bright red appearance with dark red papillæ standing out prominently. The dry, fissured, dark, coated tongue covered with sordes is rarely seen in the child and can be much more readily prevented by mouth disinfection in the child than it can be in the adult.

Nervous Symptoms.—The nervous symptoms of the child differ markedly from those of the adult; they are much less common and much less severe; the low muttering delirium and the profound stupor are rarely observed. In the vast majority of cases the nervous symptoms are confined to headache, restlessness, irritability, apathy and perhaps a tendency to somnolence, mild delirium, and transitory delusions. It should, however, also be noted that the following nervous symptoms, although very unusual,

may occur: convulsions, stupor, meningism, neuritis, hemiplegia, aphasia, melancholia and acute mania.

Respiratory Tract.—*Epistaxis,* one of the early symptoms of adult typhoid, is rare in the child.

A mild *bronchitis* is common in typhoid fever. *Broncho* and *lobar pneumonia* are serious, but rare complications.

Urine.—The diazo reaction of Ehrlich occurs in 60 to 80 per cent. of the cases; it makes its appearance about the end of the second week and increases in intensity while the disease is at its height. Its value as a diagnostic sign is impaired by the fact that it is found in measles, tuberculosis, malaria, meningitis, pneumonia, and some of the other acute infections; it is absent, however, in influenza. Acetonuria may occur. Acute nephritis is rare, but a trace of albumin is not uncommon.

Blood.—Simple anemia gradually develops with a like reduction in corpuscles and hemoglobin. The leukocytes progressively diminish in number; in severe cases they may be reduced to 2,000. The differential count shows a relative increase in mononuclears and decrease in polymorphonuclears and eosinophiles. An increase in leukocytes to over 10,000 indicates some form of septic or inflammatory complication.

**The Gruber-Widal Reaction.**—This is the most valuable of all signs or symptoms in the diagnosis of typhoid fever. It is perhaps less accurate than blood cultures, but the simplicity of its technique, bringing it within the scope of the general practitioner, gives it a value in diagnosis which perhaps will never be obtained by blood cultures. This reaction depends upon the fact that the defensive mechanism of the body, in its antagonism to typhoid bacilli, produces certain substances called agglutinins, which have the power of agglutinating and rendering motionless the typhoid bacillus. If the blood of a typhoid fever patient, containing these agglutinins, be combined with a bouillon culture of typhoid bacilli, it will readily clump them and stop their motion. When this occurs the test is said to be positive. It is commonly made under the microscope, but macroscopic tests have also been devised. The Gruber-Widal reaction is perhaps a more valuable diagnostic sign in children than in adults, as it occurs earlier in the disease and in a larger percentage of cases. In the average it may be said to occur in 95 per cent., and is commonly found as early as the seventh or eighth day. This reaction continues for a long time after the patient has recovered from typhoid, so that a positive Widal may date from a previous attack. In estimating the diagnostic value, therefore, of this test, care must be taken to determine whether the patient has ever had typhoid fever; with this excluded a positive Widal associated with an otherwise unexplained acute febrile condition justifies the diagnosis of typhoid. A positive Widal reaction may occur in jaundice, but so rarely in other conditions as to be practically negligible. A negative Widal reaction does not necessarily preclude the diagnosis of typhoid, as the examination may have been made before the agglutinins have developed in sufficient quantities to produce a reaction, or the case may belong to that

small percentage in which the reaction never occurs. Blood cultures nearly always give definite information of the existence of typhoid before a positive Widal can be obtained. At the present time, however, this method of diagnosis is largely confined to hospital practice. In this examination a bouillon culture material is inoculated with blood obtained from a vein in the arm and after twelve or twenty-four hours is examined for typhoid bacilli. As the laboratories in our cities become better equipped and the technique of the operation simplified, this method of diagnosis may become more generally used in private practice.

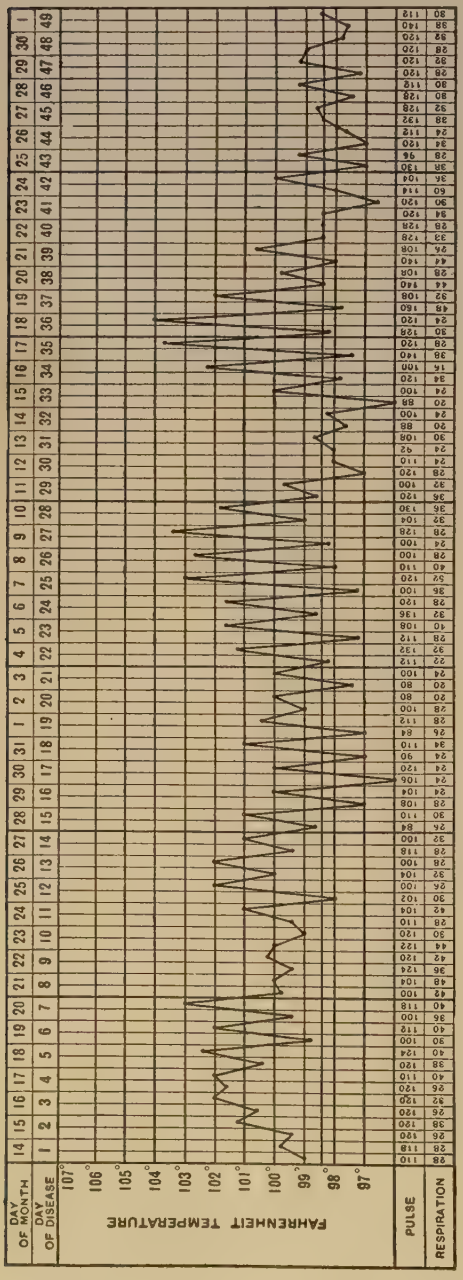



Fig. 45.—Typhoid Fever with Relapses; Child Three Years of Age.

**Relapses.**—Relapses occur in from 10 to 15 per cent. of the cases; Blackader reports fifteen relapses in 100 cases; second and even third relapses may occur. Comby reports a case with six relapses lasting in all four months. They may occur about the time a normal temperature has been reached; there is, however, usually an intervening afebrile period of from five to ten days. The relapse commonly runs a shorter and milder course and is attended with little danger.

**Complications.**—Parotitis may occur during the second or third week and may result in abscess of the parotid gland; careful mouth disinfection diminishes the frequency of this complication. Furunculosis, otitis media, pneumonia, deep-bone abscesses, arthritis and meningitis may occur, and a latent tuberculosis may become active. Within the last two years I have seen two cases of

typhoid meningitis, both of which recovered. The symptoms of meningitis in both cases developed late in the disease, and typhoid bacilli were obtained in pure culture from the cerebrospinal fluid.

**Prognosis.**—The prognosis of typhoid fever in children is much better than it is in adults. In 100 cases Blackader had only one death. Crozer Griffith reported a mortality of 3 per cent. These estimates, however, are much below the average mortality, which ranges in the neighborhood of 6 per cent. In very young infants the mortality is great, from 20 to 40 per cent.; between two and ten it is low; in children over ten it gradually increases.

**Differential Diagnosis.**—Paratyphoid fever very closely resembles mild typhoid fever, but from the standpoint of therapy there is no reason for attempting a differential diagnosis. In paratyphoid the Widal reaction is negative, but an agglutination reaction may be obtained by using paratyphoid cultures. Acute miliary tuberculosis is the disease most commonly mistaken for typhoid fever. It produces in the child a clinical picture closely resembling *adult* typhoid. In these cases, however, the absence of Widal reaction, the previous history of the child, the presence of other signs or symptoms of tuberculosis, the great irregularity of the temperature curve, the absence of rose-spots, the presence of leukocytosis and the possible finding of tubercles in the choroid should suffice to make the diagnosis. If perchance doubt still exists, the diagnosis will be made later by the long-continued fever and the complicating tuberculous meningitis or tuberculous bronchopneumonia. Intestinal grippe may present a clinical picture which suggests typhoid fever, but the accompanying catarrhal symptoms with an absence of rose-spots, Widal reaction, diazo reaction and typical temperature curve should suffice to make the diagnosis.

**Prophylaxis.**—The most important measure in prophylaxis is to use only uncontaminated water for drinking purposes, and the best safeguard in this direction is to be found in drinking *boiled water.* This precaution is necessary in most of our large cities, since the filtering systems in vogue are not an absolute protection against this disease. In country districts, when typhoid fever is epidemic, all well and spring water should be carefully avoided or should be boiled before using. As milk is also a source of danger, it is on the whole safest to use only boiled or "clean" raw milk; this is especially advisable during epidemics of typhoid fever. Oysters, known to come from beds contaminated with sewage, should be avoided. Vegetables and fruit, to be eaten raw, should be washed with boiled water. The feces and urine of typhoid fever patients should be received in a 1 to 20 carbolic acid or a 1 to 1,000 bichlorid solution. All bed and personal clothing of the typhoid fever patient should be soaked in the same carbolic acid solution for one or two hours and then boiled. The body of the patient should be cleansed following evacuation of the bowels. The nurse should exercise the greatest care in handling the excreta and clothing of the patient, and should, after such handling, carefully disinfect her hands, for only in this way may she be sure that she will not infect herself

directly, or contaminate her food supply with the typhoid bacilli clinging to her hands. While it is not necessary to isolate or quarantine typhoid fever patients, it should be remembered that the interests of the patient and the safety of the household are best served by preventing all unnecessary contact between the sick and the well.

Nursing mothers with typhoid should wean their babies, not only for the protection of the infant, but for the welfare of the mother.

Anti-typhoid inoculations with sterile typhoid cultures, as practiced by A. E. Wright in India, produce an increased temporary resistance to the typhoid infection. This prophylactic measure, however, is hardly justifiable except for the protection of hospital attendants or large bodies of people who are especially predisposed to this disease by camp life.

**Treatment.**—Typhoid fever is a self-limited disease for which we have no specific medication. It can, however, be materially influenced in its severity and shortened in its course by careful attention to hygienic details and to proper dietetic and medical treatment. The patient should be put to bed, and remain there for at least one week after all acute symptoms have disappeared. The room which he occupies should be large and well ventilated. A comfortable bed should be provided with a smooth hair mattress resting on box springs, and a rubber cloth should protect the mattress underneath the sheet. Good nursing is all-important; the patient should always be under the watchful eye of a competent observer. In older children the bed-pan and urinal should be used and it should be the object of the nurse to tactfully keep the patient as quiet as possible, allowing him to do nothing for himself that can be done by others. With a young child it may be necessary to lift him out of bed and hold him temporarily in arms; this may relieve the nervous irritability and fretfulness. The hips and back of the patient should be rubbed once or twice a day with alcohol and the position of the body changed from time to time; this is necessary in severe cases to prevent the formation of bed sores. Careful records of pulse, temperature and respiration should be taken at regular intervals and the nurse instructed as to the warning symptoms of hemorrhage and perforation, so that she may summon medical assistance at once if these symptoms occur. The mouth, throughout the disease, should be carefully cleansed three times a day with a mild alkaline antiseptic; this will greatly diminish the danger of parotid infection and prevent the dry and fissured tongue which comes from mouth contamination.

Routine Treatment.—*Diet.*—The dietetic treatment is all-important. In the child, even more than in the adult, milk is the most important article of diet. In young children, however, it is advisable to combine it with a cereal gruel. If diarrhea occurs with curds in the stool, it is advisable to substitute skimmed milk for whole milk; if this does not correct the trouble the skimmed milk or the whole milk may be peptonized and combined with barley water. It is of the greatest importance that the intestinal discharges of the child be inspected from day to day, for the

purpose of determining whether the food is undergoing abnormal fermentation and thereby adding the symptoms of intestinal toxemia to those of typhoid; in this event the dietetic measures previously outlined for the treatment of acute diarrheal diseases are applicable. The "buttermilk mixture," thick cereal decoctions, fresh meat juice and some of the proprietary meat preparations, such as liquid peptonoids, panopeptin, and Valentine's meat juice, are of value in many of these cases. The proprietary milk foods, such as malted milk and Nestlé's food, may be used and flavored with cocoa to make them more palatable. It is most important that the child should not be fed too frequently or too much, but, if possible, a sufficient number of calories should be given to satisfy nutritional demands and prevent great loss of weight. Where the digestive capacity of the child, however, is such that it must necessarily be greatly underfed, then alcohol should be used not as a stimulant but as a food to make up the deficiency in calories. Alcohol may be given in the form of the proprietary meat preparations above mentioned or good whiskey and brandy well diluted may be used. For a child six years of age, two teaspoonfuls of whiskey as a toddy or combined with carbonated water may be given every four hours; alcohol given under these conditions serves as a fuel for the cells of the body and prevents nitrogenous waste. As a part of the routine treatment the patient should be given plenty of water; this flushes the excretory organs and diminishes the toxemia. During convalescence the same diet upon which the child has gone successfully through its illness should be continued for one week after the temperature has reached normal, then gradually soft-boiled eggs, milk-toast, scraped meat-ball, orange juice, bread, and other foods may be added.

At the *onset* the patient should have a dose of calomel, followed by castor-oil or a saline cathartic. Dilute hydrochloric acid to older children and guaiacol carbonate and salol to young children may then be given as a matter of routine treatment.

The *bath* is an important part of the routine treatment; it reduces the fever, makes the patient more comfortable, quiets his nervous system, produces sleep, and acts as a general tonic. The routine treatment of tub-bathing by Brand's method, which is of great value in the treatment of adult typhoid, is neither advisable nor necessary in the treatment of this disease in children; the shock and excitement produced by the cold bath more than counteracts its good effects. In ordinary cases a tepid bath with water containing alcohol, or the fan-bath, should be given three times a day. If the fever be high and the nervous symptoms marked, this bath, prolonged for ten minutes, may be given with cool water and an ice-bag applied to the head. Where the nervous symptoms and high temperature are still more pronounced the patient may be given a cold pack by wrapping him in sheets wrung out of cold water and then covering him with a blanket; during this process the arms and legs should be rubbed to promote circulation. The character of the hydrotherapeutic measures used will largely depend upon the severity of the symptoms and the age and temperament of the

child. Whatever measures are adopted should favorably influence the symptoms and make the patient more comfortable.

Symptomatic Treatment.—It is rarely necessary to use energetic measures for the control of the *fever;* the hydrotherapeutic measures above given usually answer every purpose. Beating down the temperature does not shorten or favorably influence the disease, and medical antipyretics are therefore not indicated. The coal-tar products, such as phenacetin and antipyrin, will do more harm than good if given for any length of time.

The *nervous symptoms* can usually be controlled by hydrotherapy and an ice-bag to the head. The bromides may be of value in some cases. Opium in rare instances may be indicated in older children, but should be avoided if possible, as it aggravates the constipation and increases the intestinal toxemia. Intestinal pain, when severe enough to demand treatment, is best relieved by paregoric.

*Constipation,* which is the rule in younger children, can usually be overcome by enemata and suppositories; if, however, these do not suffice, laxatives should be used, such as milk of magnesia, castor-oil, and aromatic cascara. Much harm may be done by the constipation and resulting intestinal toxemia, and the unfounded dread of laxatives not infrequently prolongs the disease many weeks.

It should be remembered that the *diarrhea* of typhoid fever is for the most part salutary, and is nature's effort at elimination; if this symptom be not excessive it requires no treatment. From two to four loose stools in twenty-four hours is better than constipation; excessive diarrhea, however, should be controlled by subnitrate of bismuth in 5- to 10-grain doses, put up in simple chalk mixture. In aggravated cases it may be necessary to give paregoric, care, however, being taken that the diarrhea be not too suddenly controlled or converted into constipation. In every case of excessive diarrhea the diet should be modified to suit the conditions; it may be necessary to discontinue milk for a few days and substitute for it broth, albumin water, meat juice, cereal decoctions, or whiskey. When the milk is resumed it may be skimmed, peptonized, or diluted with a cereal decoction as the exigencies of the case demand. In short, a typhoid fever case with an aggravated diarrhea is to be fed as though we were dealing with an acute enteritis. If marked meteorism be present a soft rubber catheter introduced high into the colon, as recommended by Forchheimer, may be of value in carrying off gas and relieving the abdominal distention.

*Intestinal hemorrhage* requires the same treatment as in the adult, viz., absolute quiet, the patient doing nothing that can be done for him by others; temporary abstinence from food, water and ice being allowed; a hypodermic injection of morphin, 1/30 of a grain for a child six years of age, to be repeated in six or eight hours, and the application of cold to the abdomen by ice-bags, a layer of flannel intervening. If the hemorrhage be great and collapse threatens, the patient should be stimulated by hypodermoclysis of normal salt solution and by the hypodermic use of tincture of strophanthus, 2 or 3 drops, well diluted, for a child six years of age.

*Intestinal perforation* demands immediate surgical interference; when the physician suspects this condition surgical advice should be sought.

**May typhoid fever patients be sent home without additional risk?**—This is often a question of the greatest importance and one that the physician is called upon to decide. In a typhoid fever epidemic which I studied in northern Michigan some ten or fifteen patients were sent to their homes in the first and second weeks of the disease. Some of these had a fourteen hours' railroad journey, others a twenty-four hours' journey by boat, and all of them reached their homes in safety and made satisfactory recoveries. One of these patients had a temperature of over 105° F. when she was carried to the train. With these particular cases it was a question of remaining in uncomfortable summer cottages through a long illness or of being treated at home under most satisfactory conditions. Under such circumstances there should be no hesitation in sending typhoid fever patients to their homes during the first week of the disease; this is especially true of children. Other things being equal, however, typhoid fever patients should be treated where they are taken ill, and above all should not be moved in the later stages of the disease. There is more danger in traveling during early convalescence than during the first week of the disease.

## CHAPTER XXXV

### MALARIA

Malaria is an acute infectious disease caused by the plasmodium malariæ; it is characterized by more or less regular recurring intermittent or remittent symptoms, the most pronounced of which is fever.

**Etiology.**—The sole cause is the plasmodium malariæ, a hemacytozoön discovered by Laveran in 1880. This parasite is found in three forms, the tertian, the quartan, and the estivoautumnal; of these the tertian is by far the most common and is present in the great majority of the cases. On first entering the red blood corpuscle it appears as a small mass of non-pigmented protoplasm. As it gradually increases in size it becomes more and more pigmented, and the hemoglobin is gradually destroyed until the corpuscle finally appears, much paler than normal, inclosing a pigmented mass almost filling the cell. This mass splits into segments which are discharged into the blood during the chill stage; they subsequently enter other red corpuscles, where the same process is repeated. The full cycle of its development in the body is forty-eight hours, and this cycle represents the clinical manifestations of a malarial paroxysm, including the intervening quiescent period. In children, more commonly than in adults, there is a double infection by two sets of tertian parasites which mature on alternate days, thereby producing a paroxysm of acute symptoms every day. The two sets of parasites may or may not mature at the same hour on alter-

nate days, so that in these cases of double infection the acute symptoms, while they recur at practically the same hour every third day, may vary as to the time of the beginning of the paroxysms on alternate days. Another point of clinical importance is that the paroxysms caused by the two sets of parasites may markedly vary in their intensity; a severe paroxysm occurring every third day and a mild paroxysm on the intervening days. The quartan parasite, which requires seventy-two hours for its cycle of development, produces acute clinical manifestations every fourth day, and the estivoautumnal parasite, which is the cause of the irregular and more severe forms of malaria, may complete its cycle within twenty-four hours. This parasite is commonly found in the remittent malarial fevers, and while nothing like so common as the tertian parasite, is not infrequently found in the United States. The quartan parasite is rare. Malarial parasites as they occur in children present no peculiarities from the forms found in the adult. A detailed description of these various forms may be found in the text-books on practice.

**Infection.**—The anopheles, a genus of mosquito, is the all-important agent by which the disease is transferred from man to man, and so far as we are aware this is the only means by which the disease is spread. The mosquito becomes infected with the malarial parasite by sucking the blood of a malarial patient; it acts as the intermediate host for this parasite and in its body the life cycle of the plasmodium is completed. This requires about a week, and then large numbers of malarial sporozoids are excreted by the salivary glands and are transferred to man by the biting of the mosquito. In the body of the individual thus inoculated by the infected mosquito the parasites rapidly multiply, as previously described, until they are present in sufficient numbers to produce clinical symptoms. The time thus occupied in the body of their host before clinical symptoms are produced is on the average fourteen days, and this represents the stage of incubation.

In the middle and northern portions of the United States malaria is more prevalent in the late summer and early fall. It is more commonly seen in the southern and Atlantic coast states than in the north and west. It is more prevalent in the neighborhood of stagnant water and there is greater liability to contract it after sunset than during the day. All of these facts may be explained by the habits and habitats of the mosquito.

**Latency of Malaria.**—This is one of the diseases characterized by latent stages; the parasite, once it has gained access to the human body, may remain dormant or latent for long periods of time. The acute symptoms of the disease having been temporarily controlled and the patient having apparently made a satisfactory convalescence, a second attack of acute malaria may occur without a new infection. Relapses more commonly occur when the individual harboring these latent parasites has a lowered resistance occasioned by disease, or other causes which diminish the vitality and produce malnutrition, thus provoking second attacks of malaria even long after the patient has recovered from the primary attack.

**Immunity.**—One attack of malaria not only does not confer immunity, but predisposes the child to second attacks. Natural immunity is very rare. The negro, however, appears to be slightly less susceptible than the Caucasian. No age is immune. Infants, children, and adults are alike susceptible to this disease. It may even occur *in utero* and is perhaps not infrequently transmitted in this way from the mother to the child. Below is given the temperature chart of a case of congenital malaria. The mother, while pregnant with this infant, visited a malarious country and there contracted the disease. She suffered severely from tertian malaria during the last months of her pregnancy. The infant was born and lived in a section of country absolutely free from malaria, and when sixteen months of age it became violently ill with a gastroenteric infection. In the convalescence from this attack it developed a severe form of tertian malaria, the typical parasites appeared in the blood and the disease was controlled by quinin administered hypodermically. Crandall reported a case occurring eighteen hours after birth. Infant had distinct malarial paroxysms and the blood of both the mother and child contained malarial parasites. The general consensus of opinion is that infants are perhaps more susceptible but less exposed to inoculation by the malarial mosquito than adults. If fewer cases, therefore, occur among infants it is rather due to their protection from the mosquito than to the lack of their susceptibility. Löffler says that, "children are exclusively the carriers of the parasites in many districts in which malaria is endemic and that only the examination of the children will determine whether endemic malaria is present in a locality." This, he says, is an observation of Koch of great importance in the prophylaxis of the disease.

**Symptomatology.**—In children over eight or ten years of age the symptomatology is practically the same as that in adults. The periodicity of the symptom group is its chief characteristic. In the common form of malaria produced by the tertian parasite the paroxysm is usually announced by a general feeling of discomfort, associated with headache, chilliness, and sometimes a pronounced rigor, with nausea and vomiting. With the onset of these symptoms the hands and feet are cold, the lips blue, and hot water bottles and extra bed covering are utilized to make the patient more comfortable. The temperature rises rapidly and may reach 104° or 105° F. within two hours; the headache grows worse, the body chill gradually disappears, the patient becomes hot and thirsty, asks for water, and throws off the heavy covering that has been used in the stage of rigor. Soon after the temperature reaches its height it begins to fall, but not quite as rapidly as it rose. It may reach normal, or even below, in the course of a few hours, so that the duration of the fever may vary in individual cases from one to twelve hours; it commonly runs its course in four or five hours. With the fall in the temperature a profuse perspiration may occur; this symptom, however, is not so pronounced in the child as in the adult. As the temperature reaches normal the headache and other disagreeable symptoms disappear and the patient is in a condition of comparative com-

fort. The contrast between the great suffering which occurs during the height of the malarial paroxysm and the freedom from uncomfortable

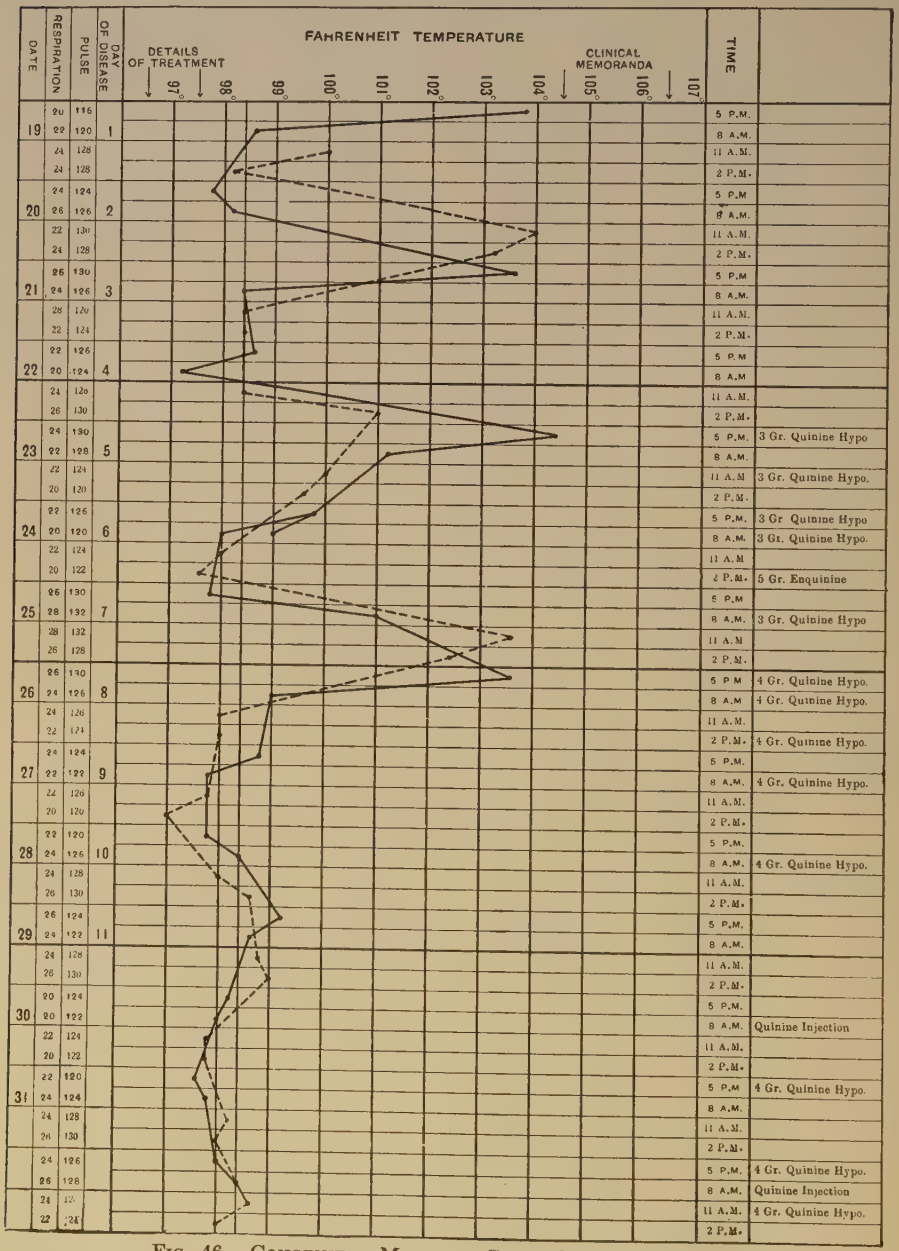



FIG. 46.—CONGENITAL MALARIA; CHILD ONE YEAR OF AGE.

symptoms which marks the interval between paroxysms is very great. The paroxysms of malaria produced by the tertian parasite commonly occur

every day, producing the quotidian rather than the tertian type of temperature. The daily paroxysms being due to a double infection by two sets of tertian parasites which mature on alternate days, thus producing a well-marked daily intermittent fever. The maximum temperature being reached at the same hour on alternate days, but perhaps varying from this hour somewhat on the intervening days. If the child be infected by only one set of tertian parasites a typical tertian intermittent fever is present; the paroxysm of fever, with its accompanying symptoms, occurring every third day and reaching its maximum about the same hour; on the intervening day the child is comparatively comfortable. The quartan parasite, which is rarely seen in the United States, produces an intermittent fever, the paroxysms of which occur about the same hour every fourth day. In the two intervening days the patient is comparatively comfortable. The estivoautumnal parasite produces an irregular type of fever commonly remittent or very irregularly intermittent. In this type of malaria great variations in the temperature may occur from hour to hour and only frequent temperature records can keep track of the excursions which the temperature may make, but on the whole, in this form of malaria, the temperature may be considered as belonging to the continuous remittent type rather than to the intermittent type.

*Spleen.*—In all forms of malaria the spleen is notably enlarged and can easily be palpated. In the typically remittent tertian form it may increase in size during the paroxysms. In chronic forms of malaria the enlargement of the spleen is very great and is an important diagnostic feature. The liver is also commonly enlarged.

*Anemia.*—Acute malaria produces a well-marked anemia which in aggravated and chronic cases may cause the characteristic malarial cachexia in which the skin has a pale yellow color. The enormously enlarged spleen which is associated with this cachectic condition may suggest the possibility of some form of severe primary anemia. A blood examination of these cases, however, shows no leukocytosis and reveals the existence of a profound secondary anemia, which is marked by a great diminution of both red blood corpuscles and hemoglobin. This secondary anemia sometimes resembles the chlorotic type.

*Intermittent neuralgia* is one of the common manifestations of subacute or chronic malaria in older children. The supraorbital nerve is a favorite site for this pain, but almost any nerve in the body may be affected. The paroxysm recurs at about the same hour every day or every second day, but is not necessarily accompanied by fever. The periodicity, however, of a neuralgia by no means classes it as malarial, since neuralgia from other causes may return at more or less regular intervals. Intermittent spasmodic torticollis occurring at the same time every day, or every second day, is a malarial manifestation rather more common in the child than it is in the adult.

*Peculiarities of Malaria in the Infant.*—From the above clinical syndrome of malaria, as it occurs in the child, there may be variations in in-

fancy. At this period the chill is absent and for it a condition of drowsiness, cold hands and feet, with marked prostration, may be substituted. Convulsions occasionally occur. Vomiting very commonly marks the onset of the paroxysm and nausea may continue until the fever begins to subside. The onset of acute symptoms is usually more abrupt in the infant and the paroxysm is sometimes associated with an acute pulmonary congestion which may suggest the onset of pneumonia.

**Diagnosis.**—The diagnosis is positively made by finding in the blood the plasmodium malariæ; this is not always a simple matter. A number of blood examinations are not infrequently required before the plasmodium is discovered. The blood for these examinations should be obtained shortly before the onset of the malarial paroxysm, when the red blood corpuscles contain the pigmented parasite. This examination should also be made before quinin is given.

Pronounced simple anemia with an enlarged spleen and regularly recurring paroxysms of fever and no leukocytosis suggests the probability of malaria. If under these conditions an examination for the plasmodium be not practicable the diagnosis may be confirmed by the specific action which quinin has on these symptoms. In tuberculosis, pyemia, septicemia, pyelitis, and other conditions we may have intermittent paroxysms of chills and fever closely resembling irregular forms of malaria. But in these conditions the plasmodium is not found in the blood and the symptoms are not specifically influenced by the giving of quinin. Probably the most common source of diagnostic error lies in the remittent forms of malaria, which may be mistaken for typhoid or some other continuous fever.

**Treatment.**—Prophylaxis may best be accomplished: First, by the destruction of the malarial mosquito (anopheles). This may be accomplished by fumigating infected houses with sulphur; destroying the breeding places of the mosquitoes by draining stagnant pools and killing the young anopheles by pouring crude petroleum over all stagnant water that cannot be drained. Second, by preventing man from being bitten by the infected mosquito. This may be done by the use of house screens and mosquito netting to cover the beds. Third, by the prompt and effective treatment of all malarial cases in the neighborhood so as to prevent the anopheles becoming infected. Fourth, by good food and proper hygiene for increasing the resisting power of the individual.

Medical Treatment.—*Quinin* is a specific for malaria. When it reaches the blood it rapidly destroys the malarial parasites and quickly terminates the symptoms of this disease.

*Method of Administration of Quinin.*—In older children the sulphate may be given in capsules. Pills are not to be used, since they may pass through the intestinal canal without being dissolved. In younger children the bimuriate or bisulphate of quinin is preferable; the solubility of these preparations promotes their absorption and thereby adds greatly to their efficacy. I have seen young children suffering from malaria who refused to yield to the sulphate and the various so-called tasteless preparations of

quinin, who promptly responded to the bisulphate given in solution or to hypodermic injections of quinin urea hydrochlorate. The following prescriptions are recommended:

℞ Quinin bimuriati .... ʒ ss
Sodii chloridi ..... grs. v
Aquæ destilatæ ..... ℥ ii

℞ Quinin bisulphat .... ʒ ss
Acidi tartaric ..... grs. xv
Aquæ destilatæ ..... ℥ ii

℞ Quinin urea hydrochlor. ʒ ss
Aquæ destilatæ ..... ℥ ii

The great difficulty that attaches to the administration of quinin in children is its very disagreeable taste. Where it is possible it is better to administer the quinin in aqueous solution, as in this form there is less possibility that it may disturb the stomach and produce vomiting. With young children a dose of the above solution may be mixed at the time of giving with a small quantity of syrup of licorice or elixir of yerba santa to cover the disagreeable taste of the quinin. Euquinin in double the dosage of other quinin preparations may be given to infants, as it is comparatively tasteless and does not irritate the stomach. The insoluble tannate of quinin put up in the form of quinin chocolates is of little or no value. It is always desirable to administer quinin by the mouth where this is possible; but an irritable stomach or failure in assimilation may make it necessary to give quinin in some other way. Under such conditions it should be used hypodermically, and the above solutions of bimuriate, bisulphate, and quinin urea hydrochlorate, when properly sterilized, may be administered in this manner. With the latter preparation I have had considerable experience. All of these are more or less irritating when given hypodermically, but the urea hydrochlorate is perhaps less so. Each injection is followed by a well-marked induration of the subcutaneous tissues which subsides in a few days. The hypodermic treatment of malaria does not, as a rule, have to be continued longer than three days. By this time the malarial paroxysms will have come under control, the nausea and vomiting will have disappeared, and quinin may again be administered by the mouth. It may also be administered by rectum; for this purpose the above solutions are available. They should, however, be largely diluted six or eight times with a dextrinized cereal decoction or with a thin starch water. There is no question but that absorption may take place when the drug is given in this manner, but the amount of absorption is uncertain and the method is far from reliable. I have little faith in suppositories of quinin and believe that they are comparatively useless. Quinin cannot be given by inunction; the drug is not absorbed through the skin. I have demonstrated this fact to my own satisfaction by careful experimentation.

*Dose of Quinin.*—At one year of age, two grains every four hours; at two years of age, four grains; at four years of age, six grains; at six years of age, eight grains; increasing one grain for every year of life thereafter. For hypodermic use the dose should be one-half, and for rectal use twice the size above given.

*Time of Administration.*—To the infant and child it is best to give quinin at regular intervals throughout the twenty-four hours when not asleep. The doses above recommended may be given at two- to four-hour intervals. When quinin is used hypodermically it should be given in rather large doses about three hours before the beginning of acute symptoms. In the older child large doses of quinin should be given four hours before the beginning of the expected paroxysm.

Apart from the quinin treatment, the management of a case of acute malaria must be purely *symptomatic.* During the chill the patient may be warmed with hot-water bottles and additional covering. Phenacetin and antipyrin in suitable doses may be given just before or at the beginning of the paroxysm to relieve the headache and make the patient more comfortable. After the fever rises and the chill disappears, an ice-bag to the head or sponging the body with cool or lukewarm water may be grateful to the patient. If constipation exists a cathartic should be given in the interval between paroxysms. A good-sized dose of calomel answers this purpose, and by many observers is believed to promote the absorption of quinin. The diet during the acute stage should be carefully selected to protect the stomach and prepare it for quinin medication. During convalescence the food should be selected with reference to the digestive capacity and nutritional demands of the child.

*Arsenic* is a very valuable remedy in convalescence from acute malaria. It acts as a blood tonic and prevents relapses. In the treatment of the chronic forms of malaria, especially those associated with enlarged spleen, malarial cachexia, and neuralgias, arsenic is of almost as much value as quinin. To young children it may be administered in the form of Fowler's solution; to older children arsenious acid may be given. Fowler's solution should be given in some palatable vehicle, such as essence of pepsin, one minim three times a day for a child two years of age and three minims for a six-year-old child. Arsenic should be administered with slight interruptions for a period of two months and during a portion of this time should be combined with some of the organic iron preparations. In younger children Fowler's solution may be combined with one of the malt and organic iron preparations. This combination is effective and palatable. In children from twelve to fourteen years of age the following prescription is of value:

℞ Acidi arseniosi ..... ½ gr.
Ferri reducti ..... 20 grs.
Quiniæ sulph ..... 30 grs.
20 capsules put up dry.
Sig. One after eating.

## CHAPTER XXXVI

### WHOOPING-COUGH

### (*Pertussis*)

Whooping-cough is an acute infectious disease characterized by a more or less violent spasmodic cough, recurring in paroxysms, accompanied by the expulsion of mucus and commonly by vomiting. The paroxysm of coughing is interrupted or terminated by an inspiratory crow or whoop, which gives the name to the disease.

**Etiology.**—The "bacillus pertussis" of Bordet and Genou is possibly the specific cause of this disease. These observers isolated this microorganism from the pharyngeal mucus, and Wollstein observed that it reacted positively to agglutination tests with the blood of the convalescent patient. At the present time, however, all that one can positively say is that whooping-cough is caused by a microorganism whose favorite habitat is the pharynx, larynx, trachea, and bronchi, and that the common exciting cause of the cough paroxysm is a plug of laryngeal or bronchial mucus. The infectious principle of whooping-cough multiplies rapidly in its human host, and is also capable of affecting dogs and cats. It is not, however, known to multiply outside the bodies of its hosts, but it may live for as long as ten days or two weeks in the dried state. It is thrown out by breathing, coughing, or sneezing; the mucus thus expelled may carry the contagion to all parts of the room and may deposit it on the clothing of the doctor or attendant, who in turn may carry it to a third party; indirect contagion, however, is, according to Morse, a very rare occurrence. The disease is usually communicated by the well coming in close intercourse with the sick, in homes, at schools, children's parties, and other public gatherings. There are unusual opportunities for the sick coming in contact with the well and thus spreading this disease, since during the catarrhal stage, when it is most infectious, the diagnosis is not usually made and the child is not ill enough to prevent its mingling with other children in the usual pursuits of life. Quarantine regulations are, therefore, ineffectual in preventing the spread of whooping-cough, which is endemic in all of our cities and which becomes more or less epidemic every two or three years. Epidemics occur throughout the year; cold weather, however, increases the number of cases and the frequency of complications. There is great variability in the virulency of different epidemics; the disease may prevail in either a mild or a severe form.

**Age.**—The majority of exposed individuals contract whooping-cough, but susceptibility is not so general in this disease as it is in measles. Nursing infants under six months of age are comparatively immune; the disease, however, may occur even in the new-born. It is most common from the end of the first to the beginning of the fifth year of life. Fifty per

cent. of the cases occur under two years of age, so that the second year of life is by far the most susceptible period. After the fourth year there is a gradually diminishing susceptibility. Old age is not exempt; I knew a physician who at the age of sixty-five contracted whooping-cough after having been repeatedly exposed over a period of thirty-five years to the contagion of the disease.

Neurotic children are perhaps not more susceptible, but they have this disease in a more severe form. Tuberculous children also have whooping-cough very severely; the disease aggravates the lymph-node tuberculosis, and the tuberculosis, on the other hand, by enlarging bronchial lymph nodes, causes pressure on the laryngeal nerves, which may prolong the spasmodic stage of whooping-cough for many months.

**Period of Contagion.**—The catarrhal stage of whooping-cough is very much the most infectious and it is especially during this period that the disease is spread. It is, however, also contagious during the spasmodic stage, and I have been much impressed with the fact that the contagion largely disappears very early in this stage. I have again and again seen children during this period of the disease brought into contact with other children in their outdoor play without spreading the infection. A quarantine lasting longer than five weeks is unnecessary. Second attacks of whooping-cough are extremely rare. The immunity conferred by an attack is as safe and as lasting as it is in any other contagious disease.

**Incubation.**—This period is rather uncertain; it probably lasts from six to ten days. Cases are reported where the catarrhal symptoms have begun within the first thirty-six hours after exposure.

**Symptomatology.**—The symptoms are divided into three stages, the catarrhal, the spasmodic, and the convalescent.

Catarrhal Stage.—The catarrhal stage begins with bronchitis, pharyngitis, and rhinitis; the pharynx, nose, throat, and eyes are usually congested. The cough is the most important symptom; it soon becomes very irritating and harsh and is associated with the physical signs of an ordinary bronchitis of the larger tubes; it is, however, more irritating and harassing than the cough of ordinary bronchitis and is worse at night; in the beginning it is not, as a rule, paroxysmal, but it is so hard and persistent that the child's face becomes congested. In infants the cough may become paroxysmal during the first two or three days of the disease. In older children the duration of the catarrhal stage differs greatly, but usually during the second week the cough, which has continued to grow worse, becomes more paroxysmal, and the typical cough of the spasmodic stage gradually develops. There is a slight rise of temperature, 101° to 102° F., accompanied by headache and general nervous irritability. With the change in the character of the cough the fever and catarrhal symptoms subside, and the general condition of the child improves.

Spasmodic Stage.—This begins at the end of the first or second week and is characterized by a more or less violent spasmodic cough, which re-

curs in paroxysms and is commonly interrupted or ended by an inspiratory whoop; mucus is expelled and vomiting frequently occurs. The cough, during this stage, recurs in distinct paroxysms, with longer or shorter intervals of rest, and the child, notified of the approach of the cough by pharyngeal irritation, places itself in a position to withstand the approaching attack. The cough is violently explosive in its character and the explosive expirations come in such rapid succession that after a time respiration almost or entirely ceases; this is followed by a loud sighing, whooping inspiration, accompanied, as a rule, by the expulsion of a mass of frothy mucus or by vomiting. If the mucus plug is not removed from the larynx the attack may be immediately repeated and followed by general exhaustion and muscular relaxation. During the paroxysm the child's tongue protrudes; its face becomes red, then a darker hue, and, in some cases, almost blue or cyanotic; its eyes bulge, the conjunctival mucous membranes are congested, and the whole body is in a state of muscular and nervous tension which is aggravated by the sense of impending danger which these attacks inspire. The above description represents a severe paroxysm of whooping-cough. These attacks may be much milder or they may be more severe and complicated with convulsions and other profound nervous symptoms. More or less emphysema occurs in nearly every severe case; it is especially marked in the apices of the lungs and in rare instances the lung may rupture, producing pneumothorax or a subcutaneous emphysema. Nose-bleed and conjunctival hemorrhages frequently occur; the latter produce the blood-shot eye so commonly seen in whooping-cough. Hemorrhages may also occur from the throat, the bronchi, and the ear; in rare instances the ear-drum may be ruptured. Incontinence of urine and of feces, especially in young children, may occur during the attack. A grayish-white ulcer may develop on the frenum of the tongue from injury to and subsequent infection of this organ. Hernia and prolapse of the rectum may result from increased abdominal pressure. Attacks may be brought on by fits of anger, excitement, and violent exercise. They may vary in duration from one to fifteen minutes, depending upon their severity. The duration of the spasmodic stage may vary from two weeks to two months. Enlargement of the bronchial lymph nodes from a complicating tuberculosis may prolong the paroxysmal cough for many months. Attacks of influenza and bronchitis may bring back the paroxysmal cough months after the child is apparently convalescent. This recurrence is strongly suggestive of bronchial lymph-node tuberculosis.

During the CONVALESCENT stage, which lasts two or three weeks, all the acute symptoms rapidly subside. The bronchitis ceases, the characteristic cough loses its whoop, becomes much milder, less paroxysmal, and gradually disappears.

BLOOD.—In whooping-cough the lymph nodes are everywhere enlarged, especially in the neck and peribronchial region. To correspond with this lymphatic irritation there is a well-marked leukocytosis, varying from 20,000 to 40,000; all forms are increased, but the lymphocytes are especially

so. The lymphocytosis is both relative and absolute and begins before and continues through the paroxysmal stage.

Urine.—Slight albuminuria may occur, but acute nephritis is a rare complication. The urine may contain sugar and an excess of uric acid.

**Course.**—Whooping-cough is a self-limited disease running its course on the average in from six to eight weeks. The whooping-cough paroxysms, however, may be continued much beyond this period from enlargement of the bronchial lymph nodes. The lymph node enlargement in these cases is commonly tuberculous. In all cases in which the whooping-cough paroxysms continue beyond the eighth week tuberculosis should be suspected, and the treatment should be the same as elsewhere given for lymph node tuberculosis.

**Complications.**—Bronchopneumonia is the most serious of all the complications and is more frequent and more dangerous in infancy than in

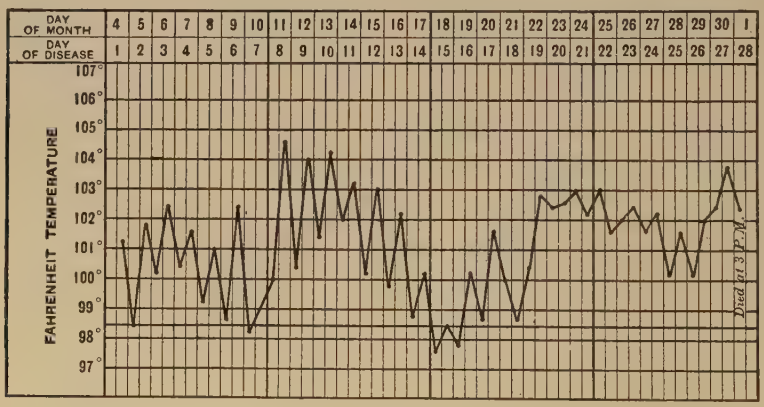



Fig. 47.—Pertussis and Measles Complicated by Bronchopneumonia.

childhood. It is produced by streptococci, staphylococci, and pneumococci, and occurs much more frequently during the winter months; it is more commonly seen in hospitals and tenement houses than in well-appointed homes. Enterocolitis is a much dreaded complication of whooping-cough *in infants* during the hot months and is responsible for no small part of the mortality of this disease. Tuberculosis is a common complication. A latent or slightly active lymph-node or pulmonary tuberculosis may be greatly aggravated into serious or even fatal forms of tuberculosis. Cardiac dilatation and weakened heart muscle are common complications and sequels of severe whooping-cough; they are manifested by shortness of breath and by an irritable and rapid pulse which is easily accelerated by exercise. Many months may be required for the heart muscle to regain its normal tone. Measles, diphtheria, and scarlet-fever may, especially in hospital practice, occur as complications of whooping-cough and add much to the gravity of the prognosis.

**Diagnosis.**—The diagnosis in the catarrhal stage is very difficult and, in

the great majority of instances, the true character of the disease is overlooked until the characteristic paroxysmal cough develops. When the disease is epidemic, or when it occurs during the summer months, its presence may be suspected early and a blood examination may confirm the diagnosis. Enlarged bronchial lymph nodes may produce a symptom group resembling whooping-cough. This condition is nearly always tuberculous, and may be differentiated by its chronic character and by the signs and symptoms of bronchial lymph-node tuberculosis. Adenoid vegetations may cause a paroxysmal cough, which is, however, not usually associated with the whoop and the after vomiting and is not aggravated at night.

**Prognosis.**—The younger the child the more dangerous the disease. Under one year of age the mortality is great and during the second year it continues high; after that it gradually diminishes, so that in private practice the disease is attended with little danger in children over four years of age. The mortality is greater during the winter months, when children are housed and ventilation is bad, and is due largely to bronchopneumonia and tuberculosis. The death rate of whooping-cough is greatly increased among the poor during the summer months by a complicating enterocolitis. W. W. Johnston called attention to the fact that statistics showed that more children died from whooping-cough in the United States than from scarlet-fever, and he estimated that 100,000 children die from it in every decade. Statistics in European countries also show that whooping-cough is scarcely second to any of the ordinary acute infections in its mortality records, and yet this disease is treated lightly and is little dreaded by the laity.

**Prophylaxis.**—A child with whooping-cough should be carefully isolated from other children, especially during the catarrhal stage. Absolute isolation during the paroxysmal stage is not consistent with the best forms of treatment, and is not in the vast majority of instances practicable. The greatest possible effort, however, should be made by the physician to protect children under three years of age, and tuberculous children of all ages, from coming in contact with the contagion of whooping-cough. It may be possible for the well to occupy the same playgrounds with whooping-cough patients who are in the late paroxysmal stage of this disease, provided this is done under proper supervision, but well children should not be allowed to enter a room that has been occupied by a whooping-cough patient until that room has been thoroughly disinfected.

**Treatment.**—The HYGIENIC TREATMENT of this disease is most important. The number of paroxysms of whooping-cough will depend largely upon the contamination of the air in which the patient lives. If kept indoors in ill-ventilated apartments, breathing bad air contaminated with dust, germs, and carbonic acid, the paroxysms of whooping-cough will be greatly increased in number, aggravated in severity, and the pulmonary and intestinal complications will be more frequent. The most important part of the treatment, therefore, is to furnish the patient with the purest air possible, both by day and by night. During the spring, summer, and

fall months it is a comparatively easy matter to keep the patient out of doors a great portion of the time without causing excessive fatigue. During the winter months the child should, on good days, spend a portion of the time out of doors and while indoors should live, day and night, in well-ventilated rooms. Where there is a family history of tuberculosis it is advisable to seek a milder climate for whooping-cough patients during the winter months.

Children old enough to lead an active life should be carefully restricted as to exercise during the severe paroxysmal stage. Over-exertion increases the number of paroxysms of cough and throws an unnecessary strain upon a heart that is already weakened by disease. It is rarely necessary, except in severe complications, to forbid all exercise by confining the patient to bed.

Dietetic Treatment.—During the hot summer months all infants under two years of age suffering from whooping-cough should, from the very beginning of the disease, be most carefully dieted, to prevent the development of that much-dreaded complication, gastrointestinal catarrh. They should be removed from the city and sent to a place where they can get good pure air. If they are not breast-fed, the artificial food formula upon which they were living before the development of the whooping-cough should be reduced in strength and in quantity; infants, for example, nine months of age, who have been taking nine or ten ounces of food every four hours, should be given five or six ounces every three hours, and the percentage of milk in the formula diminished. If intestinal trouble develops, then every attention should be directed to its correction before it becomes a gastrointestinal catarrh; it may be necessary to peptonize the milk or to substitute skimmed milk for whole milk in the food formula. At any rate the physician should be impressed with the importance of using prompt dietetic and therapeutic measures for preventing enterocolitis in young infants suffering from whooping-cough. Careful and skilful feeding is required to maintain the general nutrition in children of all ages suffering from severe attacks of whooping-cough. Throughout the disease the child's diet should be carefully scrutinized, giving to it only such foods as fall easily within the range of its digestive capacity, and, since a full meal is one of the common reflex causes of the whooping-cough paroxysm, it is advisable that the child should be fed in smaller quantities at shorter intervals. Beef-teas and highly seasoned foods which may cause pharyngeal irritation and thus excite the cough are to be avoided. In severe cases where vomiting follows the taking of food the patient should again be fed within half an hour. Food taken ten or twenty minutes after a paroxysm of whooping-cough, whether that paroxysm be accompanied by vomiting or not, is usually retained, so that the best time to feed the child in severe cases is very soon or directly after a paroxysm. In rare instances it may be necessary to give nutrient enemata of some of the soluble meat preparations or of peptonized milk.

Psychic Treatment.—The psychic treatment of whooping-cough is of

importance in children that are old enough to be thus influenced; this is especially true of neurotic children. They should be shielded from excitement, should be taught to control their temper and, most of all, should be impressed with the necessity of trying to postpone or control whooping-cough paroxysms.

Kilmer and others have recommended a snugly fitting abdominal elastic belt surrounding the body several inches above and below the region of the diaphragm. This elastic bandage is applied over, and attached to a much wider stockinet band which is held in position above by shoulder straps. Soper recommends that traction be applied in such a way as to pull both angles of the jaw forward and downward. It is believed that these devices modify the severity of the cough and diminish the frequency of the vomiting.

Medical Treatment.—At the present time the local treatment of the pharynx is not in vogue. The literature of whooping-cough is full of advice on this subject, the details of which need not be repeated here. Formalin and cresolin vapors, made by lamps prepared for this purpose, may be used to disinfect the room. These vapors may exert a favorable influence on the whooping-cough paroxysm, but should never be used at the expense of fresh air. During winter months, in damp northern climates, where the patient is necessarily confined to his room for the greater portion of the twenty-four hours, and where ventilation is limited to the partial opening of a window, or where fresh air is obtained by removing the patient from room to room about the house, both cresolin and formalin vapors may be used to advantage in disinfecting the air that the patient breathes. Oil of eucalyptus, carbolic acid, and creosote in vapor form are also recommended as room purifiers.

Quinin internally is the most valuable remedy we have in the treatment of whooping-cough. The patient may have an idiosyncrasy which prevents the administration of quinin, and in young children, especially in infants, it should be carefully given so as not to disturb the digestive organs. This is true of every drug which is administered to modify or control the paroxysms. Whooping-cough is a self-limited disease for which we have no specific medication, and in which it is a very easy matter to do more harm than good by the administration of drugs which disturb the gastro-intestinal organs, weaken the heart, tighten the cough and prevent easy expectoration. A drug, therefore, that is of real value, such as quinin, should be properly used and not abused. In children old enough to take capsules, the sulphate of quinin should be given in from 3- to 5-grain doses three times a day. In younger children euquinin should be used; this may be given in 2-grain doses at two years of age, and 3-grain doses at three years of age. I am a firm believer in the efficacy of the quinin treatment of whooping-cough and employ it in all cases where it is possible, from the beginning to the end of the disease.

As a routine treatment belladonna in some form may be administered. Jacobi, for many years, has believed this drug to be our most valuable rem-

edy. It is best given in the form of the tincture, in doses of from 1 to 3 minims, depending upon the age of the child, and should be repeated three or four times in twenty-four hours. If the severe paroxysms are not in any way modified, the dose is to be slightly increased day by day, until its physiological effects are shown in the dilated pupils or the flushed face. Bromid of potash is a valuable remedy for modifying the paroxysms. For a child two years of age the dose should be 3 grains every four hours, increasing it 1 grain for every year of life. The bromid of potash and the belladonna may be combined in the same prescription. In whooping-cough, more than in almost any other disease, medicines should be given only when they are positively indicated, but in cases requiring treatment the ordinary routine should be quinin three times a day, and bromid of potash with tincture of belladonna in a proper vehicle four times a day. Antipyrin, in from 1 to 4-grain doses, to suit the age of the child, has a marked influence in relieving the paroxysmal cough, but its depressing action on the heart demands that it should be used judiciously and for only a short period of time. Chloral hydrate is an hypnotic of some value in severe cases; it should be given in from 2 to 5-grain doses at four o'clock in the afternoon and at bedtime. The opium preparations may be recommended in older children for the control of the paroxysmal cough. Of these, paregoric (10 to 20 drops), sulphate of codein (1/50 to 1/8 grain), heroin hydrochlorate (1/100 to 1/20 of a grain) and bimecinate of morphin (½ to 1 gtt) are the most valuable. These preparations, however, have no field of usefulness in the treatment of whooping-cough in children under two years of age. A record of the number of paroxysms occurring in twenty-four hours should be kept as an indication of the value of any form of treatment. Tincture of strophanthus and tincture of digitalis may be indicated in those cases where the pulse is weak and the heart muscles dilated. The vaccine treatment of whooping-cough has not been notably successful. When whooping-cough occurs in a child in whom there is a suspicion of latent tuberculosis, the child should be actively treated for the latter disease, as outlined under the Treatment of Glandular Tuberculosis. Pneumonia, the most dreaded complication, is to be watched for and treated on the appearance of its earliest symptoms. If one makes the mistake of beginning the treatment for tuberculosis or pneumonia when these complications are not present, no harm is done, while, on the other hand, the patient's life may be lost by beginning the treatment too late.

## CHAPTER XXXVII

### DIPHTHERIA

Diphtheria is an acute infectious disease produced by the Klebs-Löffler bacillus. It manifests itself by the development and growth of a grayish-white membrane, usually located in the throat and air passages. In these foci the bacilli manufacture a very potent constitutional poison, the absorption of which is responsible for the toxic symptoms of this disease.

**Etiology.**—The Klebs-Löffler bacillus, which is the specific etiological factor in every case of diphtheria, is rod-shaped, from 2 to 4 $\mu$ in length and from 0.4 to 0.8 $\mu$ in width. It may be straight or slightly curved and clubbed at the ends. It grows readily in common culture media, but best on Löffler's blood serum, showing within twelve hours a grayish-white growth. It is non-motile, aerobic and does not liquefy the blood serum. It grows most rapidly at the temperature of the body, in a neutral or slightly alkaline media; acids and strong alkalies inhibit its growth. It is gradually destroyed by the action of sunlight and quickly killed by a temperature of 136°F. Cold inhibits, but does not kill it. It may live for months in a dried state in clothing, bedding and carpets, and the disease may be spread by the belongings of the sick room and the clothing of the attendants. It is not uncommonly found in the throats of healthy individuals, who may act as carriers of the disease to others with more susceptible mucous membranes. In milk it lives and multiplies slowly, and epidemics may be produced in this way. W. H. Park found that guinea-pigs, chickens, birds, cats, rabbits, dogs, cattle and horses were susceptible to this disease.

*Human intercourse* is the great cause of its spread. The contagion lies in the discharges which come from the local foci, usually located in the throat and nose. This bacillus is not readily carried through the air, and closer contact with the sick is necessary for the spread of this disease than for many other contagions, such as measles, small-pox and scarlet fever. Crowded tenements, schools and children's parties, which bring into intercourse the sick with the well, are important factors in the spread of diphtheria, since the carriers of this infection are very common among those who are apparently well.

Diphtheria is rare in young and nursing infants. This immunity is probably due to immune bodies derived from the placental blood and is continued by nursing milk from the breast of an immune mother. It is most common between the second and sixth year of life; after this, susceptibility gradually diminishes, until in the adult it is comparatively rare. One attack confers immunity, but this is temporary and may last but a few months. Second attacks following rather closely upon the previous attack are comparatively mild. Catarrhal conditions of the throat and nose and chronic disease of the tonsils and adenoids are predisposing causes. Diph-

theria is more common in winter than in summer. It is more prevalent in cities, but it occurs very unaccountably at times in epidemic form in remote country districts.

**Pathology.**—Diphtheria is primarily a local disease; its symptoms are largely due to the action of toxins. In prolonged cases more or less general infection with the diphtheria bacillus may occur. In these cases bacilli may be found in the blood, lymphatic tissues, liver, kidneys and other organs. The local lesions on the tonsils, soft palate, and uvula, or in the nose, larynx, and other respiratory passages, consist of an inflammation of the mucous membrane, which becomes hyperemic, swollen and infiltrated with cells. This is followed by the appearance of a grayish-white, sometimes brownish, pseudo-membrane, first occurring in patches and then spreading and forming a more or less continuous covering for the part affected. The severity of this process may vary from a mild pseudo-membranous sore throat to a necrosis, subsequent sloughing and destruction of the parts affected. An early myocarditis with acute cardiac dilatation may occur, or slower changes may take place in the heart muscle, producing a fatty degeneration of the muscle fiber, with a resulting replacement fibrosis and infiltration with small round cells. In the nerves we may have degenerative processes, both parenchymatous and interstitial, which completely destroy their function. The sensory as well as the motor nerves may be affected. In the anterior horns of the spinal cord hemorrhages and degenerative changes may occur. If the disease is prolonged sepsis becomes a part of the pathological process, and may manifest itself by the ordinary lesions of a general septicemia or septicopyemia.

**Incubation Period.**—This lasts from one to four days, and during this period there are no symptoms.

**Symptomatology.**—The ORDINARY FORM of diphtheria begins with a sore throat. The tonsils and pharynx are swollen, congested, and present one or more grayish-white patches which coalesce, forming a pseudo-membrane that gradually takes on a grayish-brown color. It may spread over the tonsils, uvula and pharynx, and may extend into the nose or through the larynx down the trachea into the bronchial tubes. Hand in hand with the extension of this membrane the local inflammatory conditions in the throat are increased, so that the patient may complain more and more of difficulty in swallowing. The odor from the breath may become fetid. The lymph nodes at the angle of the jaw, which are always enlarged, may become more and more swollen, involving the intervening connective tissue, producing a doughy-like swelling in the region of these glands. The pseudo-membrane may not only be seen in the locations described, but as the disease progresses it may be coughed up or discharged from the nose, and with the breaking loose of these membranes hemorrhages may occur. In mild cases the above local symptoms are very much modified, so much so that the patient may scarcely be conscious that he has anything more than a very mild tonsillitis; in the severe cases they may be greatly aggravated by the necrotic and gangrenous processes which occur.

There may be headache, backache, general malaise, and an early rise of temperature reaching in the first twenty-four hours 102° to 104° F. The fever, as a rule, commences to fall within twenty-four or thirty-six hours; in uncomplicated cases it may reach normal within a few days. A secondary rise is due to some complication, usually of septic origin; when this occurs the temperature runs the irregular course of septicopyemia. The temperature may vary greatly; in the most virulent cases it may be normal or even subnormal, so that from a diagnostic, as well as a prognostic, standpoint it may be very deceptive.

The general prostration of the patient during the first twenty-four

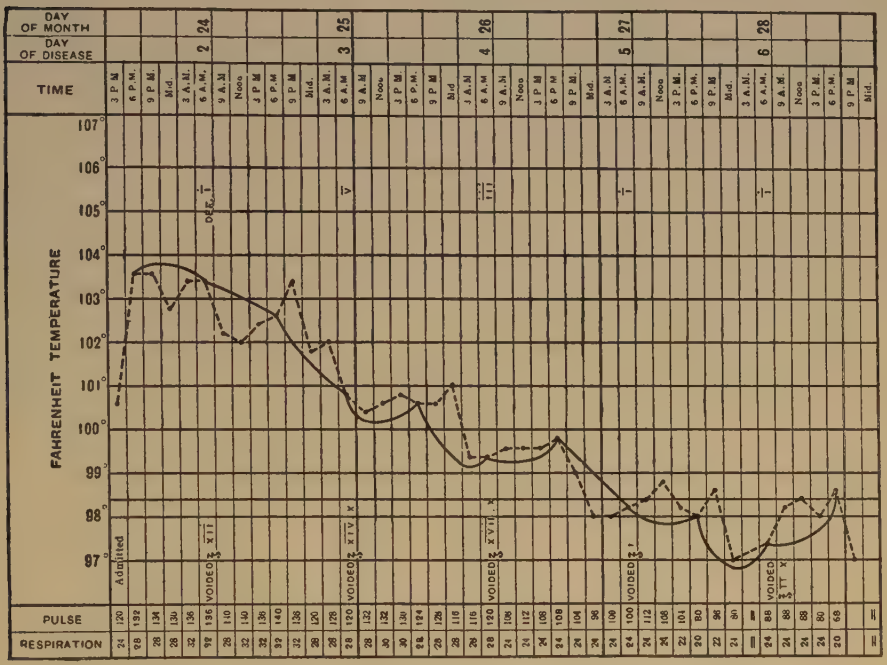



Fig. 48.—Pharyngeal Diphtheria Treated with Antitoxin.

hours of the disease is an important prognostic sign. The more profound the prostration the more severe the intoxication. In the most violent cases the patient is almost at once overwhelmed by the toxemia. Extreme prostration, high fever, rapid, feeble, irregular pulse with profound nervous symptoms, such as stupor, coma and convulsions, may be associated with hemorrhages from the mucous membranes, and with a purpuric rash over the body. These foudroyant cases, which may die within the first two days, are fortunately very rare.

The blood presents no characteristic changes. There is a simple anemia and a marked leukocytosis, sometimes as high as 48,000. The polymorphonuclears are usually increased.

Laryngeal diphtheria may be primary or secondary; fatal cases

are seen where the throat and pharynx present no evidence of a pseudo-membrane. A slight pseudo-membrane behind the pillars of the fauces may have been overlooked, but it is well to keep in mind the fact that laryngeal diphtheria in rare instances occurs with little or no preceding evidence of sore throat. The membrane, however, in a great majority of the cases extends from the throat into the larynx, and the symptoms of laryngeal diphtheria are therefore commonly preceded by some evidences of tonsillar or pharyngeal diphtheria. In extremely rare instances it is believed that the membrane may occur primarily in the trachea or bronchial tubes and ascend to the larynx. The diagnosis of tracheal or below-the-

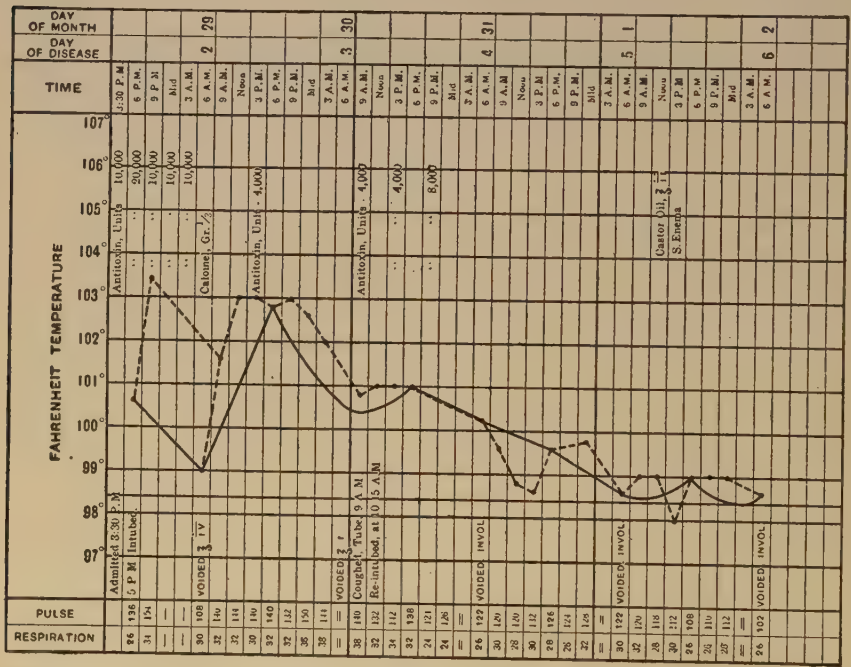



Fig. 49.—Laryngeal Diphtheria Treated by Intubation and with Antitoxin.

larynx diphtheria is made by the coughing up of membranous casts from the trachea or bronchial tubes and by the failure of laryngeal operations to relieve the stenosis, and also perhaps by the evidence of unilateral blocking of the bronchial tubes as made out by percussion and auscultation.

The classical symptoms of laryngeal diphtheria are: a croupy cough of metallic character, increasing in severity and associated with a gradually increasing hoarseness, dyspnea, and loss of voice. The dyspnea is slightly worse at night, and slowly increases in severity day by day until the stridor and labored breathing may be heard in all parts of the room. On the third or fourth day the membrane in the larynx may cause complete obstruction and end the life of the patient by suffocation. As the breathing becomes more and more difficult, the patient sits up in bed and fights for

air. All the accessory muscles of inspiration are brought into play as the child with tremendous effort attempts to force air into the lungs; the alæ of the nose are dilated, the suprasternal notches are sunken in, the diaphragmatic groove is contracted, and the whole attitude of the patient is characteristic of air hunger. The croupy cough comes on in paroxysms, is very persistent and is only temporarily, if at all, relieved by vomiting.

Diphtheria of the Eye.—True diphtheritic ophthalmia, before the days of antitoxin, was one of the most dreaded of all the complications of this disease, and it is still much feared because it so frequently results in loss of sight. The conjunctiva is violently inflamed and covered with a white or gray membrane. There is a profuse purulent discharge and the lids are so swollen that they can be separated with difficulty. The cornea quickly becomes involved and its destruction may cause loss of vision.

Diphtheria of the Vulva.—This is a rare complication, but it may occur in infants as a result of direct inoculation. The parts are much inflamed, swollen and covered with a pseudo-membrane. These cases commonly run a benign course and terminate in recovery.

**Paralysis.**—The heart may suffer both early and late in the disease in the acute onslaught of very severe cases; asystole and diminished systole may result from splanchnic paralysis. During the second or third week vomiting, abdominal pain, cardiac gallop rhythm and irregular respiratory movements usually mean the involvement of the pneumogastric nerve. In both of these conditions there is imminent danger of cardiac paralysis. Between the third and the sixth week fatty degeneration of the heart muscle with cardiac dilatation may occur, and the heart's action may become irregular, intermittent and feeble, and a slight murmur may appear at the apex. These cases, however, commonly make a slow recovery, the cardiac weakness persisting for months after convalescence has been established. It should be remembered that a moderate degree of irregularity and disturbance of the cardiac rhythm may occur at any period of the disease without necessarily indicating serious cardiac involvement, but when the symptoms of splanchnic or vasomotor paralysis appear the life of the patient is in imminent danger.

The *post-diphtheritic form of paralysis* commonly occurs during apparent convalescence, from two to five weeks after the onset of the initial symptoms, and its appearance may confirm an uncertain diagnosis. This form commonly begins in the palate and is made evident by the nasal tone of the speech and by difficulty in deglutition; fluids taken into the mouth sometimes return through the nose. In these cases the patellar reflex should be studied as an indication of the probable extension of the paralysis; the absence of this reflex indicates approaching paralysis of the legs. This may be followed by paralysis of the muscles of the eyes, arms and trunk, gradually resulting in complete paralysis of almost all the voluntary muscles. The prognosis in this form of paralysis is favorable; a slow but complete recovery, extending over months, usually takes place.

**Complications.**—General septicemia, or a septicopyemia, are common

complications of diphtheria, and the course of the sepsis is similar to that described under Scarlet Fever. Severe cases of this character are usually associated with ulcerative and gangrenous sore throats, and with extensive cervical adenitis, involving the cellular tissue of the neck, which may or may not terminate in suppuration. This local symptom group is accompanied by profound constitutional symptoms, such as marked prostration, a septic fever or subnormal temperature, a feeble, intermittent pulse and stupor, deepening into a semi-comatose condition.

*Otitis media, mastoiditis* and *purulent infections* of the *frontal* and *ethmoidal sinuses* may occur.

*Bronchopneumonia* is the most dreaded of all the complications. It occurs most frequently in septic and in laryngeal cases; it is especially to be feared following tracheotomy and intubation for laryngeal stenosis. It is found in 50 to 70 per cent. of fatal cases. The onset is marked by an increase in the fever, cough and dyspnea, and a careful physical examination reveals either a unilateral or a bilateral bronchopneumonia.

*Nephritis,* according to Baginsky, occurs in 42 per cent. of the severe cases. It is, however, a rather uncommon complication of the mild cases, and, as this type of the disease greatly predominates, it does not perhaps occur in more than 5 to 10 per cent. of all cases; a slight albuminuria, however, is observed in about 30 or 40 per cent.

*Simple enteritis* occurs very frequently, but pseudo-membranous gastroenteritis is very rare; when present, however, it usually causes a fatal termination.

**Course and Duration.**—In simple tonsillar and pharyngeal diphtheria, the acute symptoms last from three to five days. In cases which are complicated by septic infection the disease may be indefinitely prolonged. The antitoxin treatment greatly shortens the course and modifies the severity of the disease.

**Prognosis.**—Age is a very important prognostic factor. During the first year the death rate is 50 per cent., the second year 30 per cent.; after the seventh year 7 or 8 per cent. The severity of the infection is a determining factor in the prognosis; some epidemics are very malignant, others are very mild; the character, therefore, of the prevailing epidemic may assist in determining the prognosis in an individual case. The parts affected very materially influence the prognosis; in laryngeal and tracheal diphtheria the death rate may reach 60 to 70 per cent.; in severe nasal diphtheria, with a complicating sepsis, the prognosis is also serious; in simple tonsillar diphtheria the prognosis is good. The previous condition of the patient may determine in part his powers of resistance, and may, therefore, influence the prognosis; in feeble, malnourished children the prognosis is not as good. The treatment is the important determining factor in the prognosis. If antitoxin is given within the first twenty-four hours, less than 5 per cent. die; within the second twenty-four hours, less than 10 per cent.; within the third twenty-four hours, about 20 per cent.;

within the fourth twenty-four hours, about 40 per cent. In the laryngeal cases the death rate is reduced by antitoxin from 65 to 25 per cent.

**Diagnosis.**—For the diagnosis of this disease the physician must depend first upon his clinical observations, and second upon the bacteriological findings. The clinical diagnosis is made by the appearance of a pseudo-membrane on the tonsils, uvula, pharyngeal wall, and sometimes in the nose and in the larynx, when it is not visible elsewhere. In most instances there is nothing absolutely pathognomonic in the appearance of this membrane, but the physician comes by experience to learn that grayish-white patches of pseudo-membrane, having a tendency to spread, and located as above noted, usually mean diphtheria, and that all such cases should have antitoxin at once before the result of the bacteriological examination denies or confirms the correctness of his diagnosis. The bacteriological findings are of the utmost importance, and if made early in the disease are more reliable than the clinical appearances. But it should be remembered that they are by no means infallible; for instance, in rare cases pseudo-diphtheria bacilli may cause confusion, or again the membrane may be so located in the air passages that it is not reached by the cotton-wrapped probe from which the culture is made, or late in the disease pyogenic cocci and other microorganisms may have so replaced the diphtheria bacilli that they are not found in the culture. Clinical and bacteriological findings, therefore, should go hand in hand. When they agree, as they do in most instances, there is no question as to the diagnosis; when they disagree the patient should have the benefit of the doubt and be given antitoxin, and the subsequent history of the case will determine the wisdom of this action. From the standpoint of preventive medicine, the laboratory diagnosis is all-important. It is relied upon to determine whether a quarantine shall be established and when it is to be discontinued. Patients should not be discharged until a negative throat culture has been obtained. Paralysis of the soft palate and other post-diphtheritic paralyses may often make a late diagnosis of diphtheria in cases that were supposed to have suffered from follicular tonsillitis. The paralysis which follows diphtheria is a neuritis and is to be differentiated from infantile paralysis and cerebral palsy; this differentiation, however, is taken up under the latter disease. It is only important here to note that a paralysis which occurs with or follows a membranous sore throat and which begins in the palate is, in almost every instance, due to diphtheritic neuritis. Traumatic pseudo-membranes following operations on the tonsils, or injury to the throat or mouth from caustic alkalies, may be differentiated from true diphtheria by bacteriological findings and by the previous history and symptomatology.

*Diphtheritic laryngitis* may be differentiated from spasmodic catarrhal croup by the following points: In catarrhal laryngitis the attack of croup comes on suddenly, usually in the early part of the night; the child wakes up with a hoarse, barking cough, struggles for breath and has a laryngeal stridor that can be heard in all parts of the room. The attack reaches its height the first night; the next day the child is comparatively com-

fortable, with perhaps a little hoarseness and croupy cough; the second and third nights the attack may be repeated, but grows less severe. In laryngeal diphtheria, however, the laryngeal stridor comes on more slowly, gradually increasing in severity for three or four days, until the child's life is imperilled by strangulation; the stridor also continues throughout the day; it may be worse at night. The presence of a membrane in the throat, and a bacteriological examination, may assist in the diagnosis. An emetic, which gives such prompt relief in simple laryngitis, has little influence on the laryngeal stridor of diphtheritic croup. If necessary the administration of chloroform may be resorted to, to assist in the diagnosis; chloroform relieves the stenosis of simple laryngitis, but has little influence in laryngeal diphtheria.

**Prophylaxis.**—As a matter of general prophylaxis all children having diseased tonsils, adenoids or nasal mucous membranes should have these parts properly treated, and, if possible, put in normal condition so that they may be less predisposed to contagion. Children who have been exposed to diphtheria should be given an immunizing dose of from 500 to 1,000 units of antitoxin and should also have their throats and noses carefully douched with physiological salt solution, or with some alkaline antiseptic. In exposed institutional children the immunizing dose of antitoxin should be repeated in three weeks.

Although the radius of infection in diphtheria is more contracted than it is in most of the contagious diseases, the patient should be isolated and a rigid quarantine instituted, which should be continued until convalescence is established and until a bacteriological examination of the throat has demonstrated that it is free from diphtheria bacilli. The preparation of the sick room and other details of quarantine are described in the chapter on Scarlet Fever. In the average case of diphtheria the quarantine lasts two weeks.

**Treatment.**—In diphtheria ANTITOXIN, discovered by Behring in 1890, we have a specific remedy that can be relied upon to exert a curative influence in every case when it is given at the proper time and in the proper dosage. It is prepared from the blood serum of the horse, immunized by gradually increasing doses of the diphtheria toxin. The horse serum thus obtained contains an antitoxin which combines with and neutralizes the toxin of diphtheria, and quickly brings to its termination the localized inflammation caused by the diphtheria bacillus. This specific antitoxin came into more or less general use in 1894, following an exhaustive investigation by M. Roux of the work which had been done upon this subject up to that time. This paper was so convincing that antitoxin was at once accepted by the medical world as a specific remedy for diphtheria. From that day to the present time it has been in general use and as a result the mortality from diphtheria throughout the world has been enormously diminished. The testimony wherever this remedy has been systematically and intelligently used is so absolutely convincing as to its specificity that it is difficult to understand at the present day how there can be any opposition to its use.

The following chart from McCollom graphically illustrates the value of antitoxin in decreasing the death rate of diphtheria:

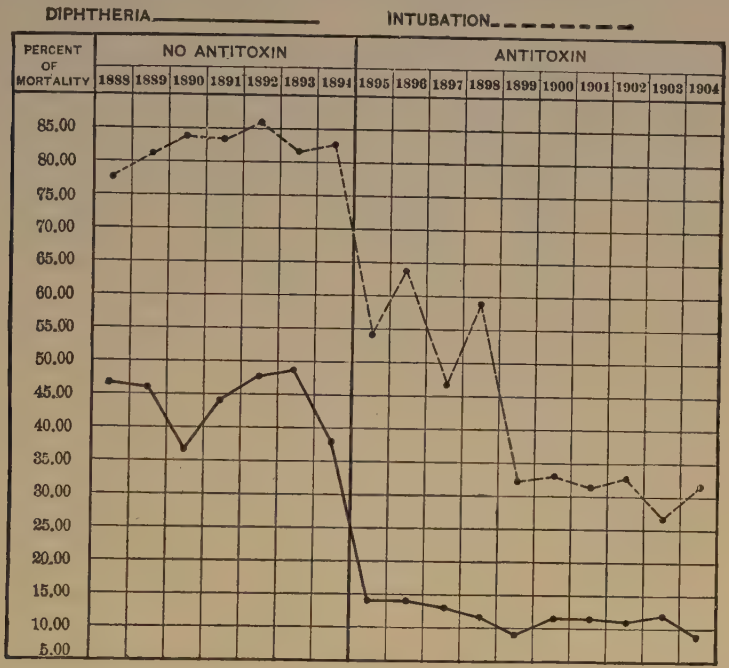



FIG. 50.—PER CENT. OF MORTALITY OF DIPHTHERIA AT THE BOSTON CITY HOSPITAL, PROPER, AND AT THE SOUTH DEPARTMENT FROM 1888 TO 1904, INCLUSIVE. PER CENT. OF MORTALITY OF INTUBATIONS FOR THE SAME TIME. 1888 TO 1894 NO ANTITOXIN. 1895 TO 1904 ANTITOXIN. (McCollom.)

Antitoxin should be given by subcutaneous injection in the loose tissue of the back below the angle of the scapula, or in the buttocks. The skin should be carefully scrubbed and disinfected, and the injection made with a sterilized syringe. At the present time the various manufacturers furnish antitoxin in sterile syringes ready for use. In uncomplicated tonsillar or pharyngeal diphtheria the initial dose should be 4,000 units, except in infants under one year of age, to whom 2,000 or 3,000 units should be given. If the symptom group be not greatly improved, a second dose should be given eight hours later. In neglected cases, coming into the hands of the physician on the third or fourth day of the disease, the treatment should be begun with 10,000 units, and this dose repeated in eight hours, if no improvement is noted. In laryngeal diphtheria the initial dose should be 10,000 units, and if the laryngeal stenosis be not relieved, from 40,000 to 50,000 units should be administered within the next three days. After a large experience with this class of cases, in the wards of a city hospital, I am convinced that the mortality in laryngeal diphtheria would be less and the number of operative cases fewer if they were treated with large doses. In diphtheria of the eye large doses of antitoxin are necessary to save the

eye; 10,000 units should be given every six hours until the purulent conjunctivitis is controlled. To a child three years of age I recently gave 70,000 units over a period of four days, with the result that the eye was saved and no untoward symptoms followed the antitoxin. The local treatment should be directed by an oculist; it consists in ice-cold applications, the frequent irrigation of the eye with a 3 per cent. boracic acid solution, and the dilatation of the pupil with atropin. If only one eye is affected, the other should be covered with a watch glass and carefully sealed with adhesive plaster and collodion to prevent its infection.

In advocating large and frequent doses of antitoxin in the cases of diphtheria which threaten life or endanger the eye, I do not wish to convey the impression that these large doses are necessary in the simpler forms of diphtheria seen in private practice. As previously noted, most of these cases recover promptly under from 4,000 to 8,000 units of antitoxin, and the giving of enormous doses unnecessarily, while it may do no harm, entails an expense which prejudices the public against the use of this most valuable of all remedies. The only unpleasant results that I have ever seen from antitoxin are the skin rashes which so commonly follow its use, and the size of the dose has little to do with the appearance of these rashes. Certain anaphylactic phenomena may occur as annoying sequels of the antitoxin treatment; the most common of these is urticaria; more rarely a rash, morbilliform or scarlatinaform in character, may appear, which in connection with the sore throat may suggest scarlet fever; arthralgia and enterocolitis are occasionally seen. It is believed that, in children of asthmatic constitution, dangerous and even fatal anaphylactic phenomena may occur. I have never seen such a case.

Local Treatment.—Before the days of antitoxin the life of the child often depended upon the thoroughness with which the throat and nose were cleansed with antiseptic sprays, gargles and douches. The throat and pharynx should be swabbed alternately with a 1:1000 bichlorid of mercury and 20 per cent. argyrol solutions in the bad cases of nasal and pharyngeal diphtheria in which the septic cocci are playing an important rôle in producing symptoms. Where the lymphatic glands of the neck are acutely inflamed an ice-bag is the best application. If this enlargement continues for a number of days poultices may be substituted for the ice and, if suppuration occurs, the abscess is to be opened.

In laryngeal diphtheria steam inhalations are of some value, and combined with this, calomel may be sublimed. The croup kettle and calomel sublimations, however, which were such important parts of the treatment before the days of antitoxin, are now but rarely used. They have a certain degree of efficacy which should justify their remaining a part of the treatment of laryngeal diphtheria; however, when the pseudo-membrane produces such stenosis of the larynx that the child is becoming exhausted in its efforts to force air into the lungs, this stenosis must be relieved either by tracheotomy or intubation, but following either of these operations the antitoxin treatment is to be continued. Of these two operations intubation

is universally recognized to be the best. It has many advantages over tracheotomy, it is bloodless and therefore not objected to by the parents, it is quickly accomplished and serves the purpose of relieving the stenosis, and is less likely to be followed by bronchopneumonia than is tracheotomy. On the other hand, intubation cases require more careful watching; for this reason, in remote country districts, where the patient cannot be under constant supervision, tracheotomy is to be preferred. It is also to be used where intubation fails to relieve the stenosis, or when the tube is frequently coughed up.

Intubation.—Dr. Joseph O'Dwyer, of New York, in 1883 perfected intubation, and the intubation set which he devised remains to-day, with unimportant modifications, in general use. The O'Dwyer intubation set consists of seven tubes made of vulcanized rubber on a metal frame; a gauge for determining the size of the tube suitable to the age of the patient; obturators and a handle for manipulating the tubes in their introduction; an extractor for removing the tubes from the larynx, and a gag for holding the mouth open during the operation. The patient is wrapped in a blanket with his arms at his sides and held firmly, sitting upright in the lap of the nurse, or placed on a table in a horizontal position with the head thrown backward; the recumbent position is to be preferred. The jaws are widely separated by the introduction of the mouth gag, which is held firmly in position by an assistant. The introducer, with tube attached, is held in the right hand, and the forefinger of the left hand is introduced into the mouth and directed downward over the tongue until the epiglottis is felt. This is hooked forward and the finger inserted into the chink of the glottis. With the tip of the finger in this position the tube is inserted into the mouth, following the line of the finger, being careful to keep it in the median line until it reaches the chink of the glottis. As it enters the larynx the introducer is removed and the tube pushed into position by the left index finger. If the tube is in proper position the symptoms of stenosis are promptly relieved. If the operation has been unsuccessful, the tube may be withdrawn by the thread to which it was attached before its introduction, and the process repeated until successful intubation is accomplished. An anesthetic is unnecessary in this operation. After a variable period of from one to six days, when the disease has come under the control of antitoxin, the tube should be removed. This operation is more difficult than inserting the tube. The index finger of the left hand is introduced as before until it reaches the head of the intubation tube. With this finger as a guide the extractor is inserted and the tube withdrawn. If the stenosis returns the tube is to be again inserted.

In skilful hands, intubation is a comparatively simple operation which can be performed with little danger to the patient. Where the pseudo-membrane is extensive the tube may push the membrane before it into the trachea, causing obstruction, which is commonly relieved by a violent fit of coughing and the expulsion of the detached membrane. If suffocation threatens, tracheotomy may be necessary to save the life of the

child. The high operation above the isthmus of the thyroid is to be preferred, since it is less difficult and is attended with less bleeding; occasionally the low operation may be necessary. Following intubation, care must be exercised in feeding to prevent the passage of food material through the tube into the bronchi; this accident produces violent fits of coughing. It was formerly believed that food entering in this way was a common cause of pneumonia, but the carefully devised experiments of Northrup have shown that there is little or no danger from "food pneumonia." In a few cases it may be necessary in giving fluids to place the

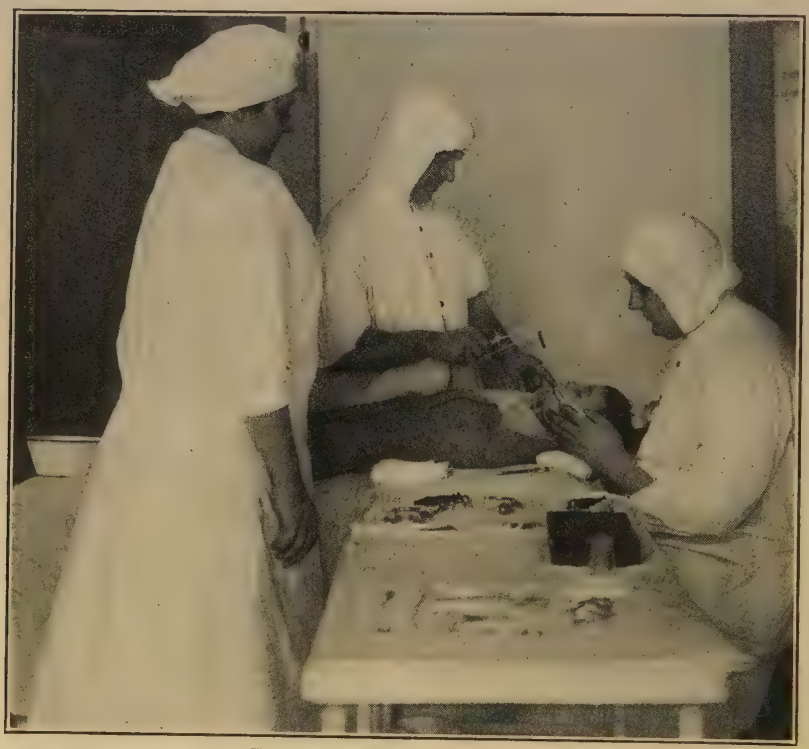


FIG. 51.—INTUBATION POSITION.

child upon its back with its head thrown backward over the side of the bed, so that the pharynx is lower than the larynx. In very rare instances it may be necessary to introduce food through a stomach tube. Bromid of potash combined with belladonna in some palatable vehicle may be of value in relieving the cough and irritation of laryngeal cases in which a tube has been inserted.

GENERAL TREATMENT.—The child is confined to bed even in the mildest cases because of the danger of cardiac paralysis. Absolute quiet should be insisted upon until convalescence is fully established. The patient throughout his illness should have fresh air night and day, and be placed under the best hygienic conditions possible. Throughout the treatment the

diet should be largely milk. Ice-cream is especially grateful to many of these cases, and it may be given freely. Cereals may also be allowed. From the beginning, albuminous foods, however, such as meat and eggs, should be dispensed with until convalescence is established. In severe and neglected cases whiskey and brandy should be given in good-sized doses, well diluted—a teaspoonful every two or three hours for a child three or four years of age. In these cases also stimulation may be necessary. Strychnin, caffein sodium benzoate, strophanthus, digitalis and normal salt solution may be given as indicated to sustain the pulse, overcome exhaustion and prevent collapse. In more desperate cases camphor and ether may be used hypodermically.

Treatment of Complications.—In the treatment of *splanchnic paralysis* previously referred to, Forchheimer says: "My remedies for vasoconstriction are adrenalin and caffein, administered hypodermically. Adrenalin especially possesses the property of producing contraction in the blood vessels supplied by the splanchnic nerve; as its effects are transitory, it must be administered frequently—every two to three hours in the 1:1,000 solution, of which from 1 to 1.5 c. c. are given at a dose. Caffein sodiosalicylate may be given, combined with adrenalin or alone; when the patient begins to improve, caffein alone is given (v. Chronic Myocardial Insufficiency), the adrenalin being gradually discontinued, because I have found that the action of caffein is more lasting than that of adrenalin. Further, I apply two or three ice-bags to the abdomen, to act upon the abdominal reflex, because this increases blood pressure and reduces frequency of systole."

*Septicemia* becomes a part of the pathological process in untreated cases by the third or fourth day. These cases should therefore be treated not only for diphtheria, but also for septicemia. If they do not yield readily to large doses of diphtheria antitoxin, then anti-streptococcic serum and inunctions of unguentum Credé should be given (see Scarlet Fever). The latter is especially indicated in severe inflammation of the lymphatic glands of the neck.

*Bronchopneumonia* is to be treated as outlined under the treatment of this disease, except that the onset of a pneumonia complicating diphtheria is an urgent indication for large and repeated doses of antitoxin. The pneumonia in these cases is caused by the extension of the pseudomembrane into the smaller bronchi, and any treatment other than antitoxin is of little avail.

*Post-diphtheritic Paralysis.*—This is to be treated by large and repeated doses of diphtheria antitoxin. Comby has clearly demonstrated the value of this remedy in the cure of diphtheritic neuritis. From 3,000 to 4,000 units of antitoxin should be given daily over a period of eight or ten days. While antitoxin greatly shortens the duration of the post-diphtheritic paralysis, it should be remembered that in nearly every instance, even in the absence of this treatment, time restores the function of the nerve and cures the paralysis. In addition to the specific treatment with

antitoxin post-diphtheritic paralysis is to be treated as outlined in the chapter on Neuritis. Good food, fresh air and proper hygiene are important, and following the acute symptoms general massage and electricity are of advantage.

## CHAPTER XXXVIII

### INFLUENZA

Influenza is an acute infectious and highly contagious disease caused by the influenza bacillus. It is characterized by fever and acute catarrhal processes, especially of the respiratory passages.

**Etiology.**—The influenza bacillus, discovered by Pfeiffer in 1892, is the exciting cause. The bacillus septus, the pneumococcus, the micrococcus catarrhalis, the pyogenic cocci and other microorganisms may produce symptom groups characterized by catarrh of the respiratory passages which cannot easily be differentiated from influenza. These cases are spoken of as common colds or epidemic coryzas and are to be treated in the same manner as true influenza.

The Pfeiffer bacillus is found in great numbers and almost in pure culture in the mucous discharges from the nose, throat and bronchi in the early stages of influenza; later it is associated with streptococci, staphylococci and pneumococci, which are found not only free but within the pus cells. It is also commonly found in catarrhal discharges from the respiratory passages where there is an entire absence of ordinary acute influenza symptoms; such cases, it is believed, represent a localized chronic phase of this disease, and are associated with inflammation of adenoids, bronchitis or apex catarrh of the lungs. Influenza bacilli are also associated with pyogenic organisms in chronic pus forming processes such as otitis media and infections of the sinuses of the face. The Pfeiffer bacillus rarely produces infection in lower animals. Inoculation experiments have for the most part resulted in failure.

The specific cause of this disease is commonly spread by coughing, sneezing and expectorating. The danger lies not only in the moist bacilli thus discharged, but in the dried bacilli which may contaminate public conveyances, homes, schools and other places where people are gathered together indoors. Influenza bacilli are so readily disseminated both in the dry and in the moist state that the disease is highly contagious and spreads rapidly through households, schools and communities in epidemic form. A number of pandemics of this disease have occurred; the last one in 1889, when 30 or 40 per cent. of the entire population of our large cities suffered more or less from it. Since that time the disease has been present to a greater or less extent in all of our large cities, so that occasional cases may occur throughout the year. The influence of climate in favoring its spread is shown by the fact that it becomes epidemic during the winter months, beginning usually before the first of January, reaching a

maximum in February, and gradually subsiding in the early spring. One of the noticeable features of these epidemics is house infections, the disease persisting throughout the winter and early spring in certain houses. The children living therein suffer during this time from frequent recurring attacks of mild influenza, which are probably due to reinfection. In other instances these repeated attacks may be due to relapsing or chronic influenza, the Pfeiffer bacillus never entirely disappearing from the adenoids, tonsils, sinuses and other of their favorite hiding places.

Nursing infants under six months of age are practically immune, and the disease is comparatively infrequent during the second six months of life. It may, in rare instances, occur even in the new-born. After the first year susceptibility to this disease rapidly increases, so that children three or four years of age are almost, if not quite, as susceptible as adults. It is common even in old age. At the two extremes of life the disease, by reason of its complications, is more dangerous.

**Pathology.**—The pathological changes which properly belong to influenza are those of a catarrhal inflammation of the mucous membrane of the respiratory passages. The accessory sinuses are not infrequently involved and the tonsils, adenoids and neighboring lymphatic glands are enlarged by congestion or inflammation. The mucous membrane of the intestine may be acutely inflamed, and almost every organ and tissue of the body may be either directly or indirectly injured by the Pfeiffer bacillus and the pyogenic organisms which are so commonly associated with it in its destructive processes.

**Incubation.**—The average period of incubation is from two to three days, but it is commonly believed that this period may vary from twelve hours to a week.

**Symptomatology.**—The symptom group presented by influenza is very variable, and different epidemics may be characterized by the predominance of a certain set of clinical symptoms, which in another epidemic may be largely in abeyance. The most characteristic symptom group, however, is that produced by catarrhal inflammation of the respiratory passages.

*Onset.*—The temperature rises rapidly, in some instances reaching 105°F. within the first twenty-four hours; the younger the child the higher and the more rapid the rise of the temperature; there may be a sensation of chilliness or even a decided rigor; in infants convulsions may occur. The discomfort of this period is very acute, the head, back, and every part of the body may ache, and there is usually complete loss of appetite with more or less gastric disturbance. The prostration is marked, quite out of proportion to the other symptoms, and the child presents the appearance of being very ill. The younger the child the more pronounced are these general symptoms. The fever may begin to fall within thirty-six hours, but commonly does not reach normal until the third or fourth day. It may, however, be prolonged by various complications. In subacute or chronic forms of the disease the temperature after reaching normal may slowly rise again, and a slight and variable fever may last for weeks.

*Coryza,* which is one of the most characteristic symptoms, may occur early in the disease, but is usually delayed until the second day. Tonsillitis and pharyngitis as a rule precede the coryza. The pharynx is markedly congested, the tonsils enlarged, and not infrequently a complicating infection produces a white exudate. These symptoms are usually followed by bronchitis or laryngitis, which give rise to an irritating cough which may be hoarse and paroxysmal in character, in some instances resembling the whooping-cough paroxysm.

*Nervous Symptoms.*—In young children the disease may commence with vomiting, stupor and symptoms of meningeal irritation, closely resembling a beginning meningitis. This profound toxemia, involving the nerve centers, while not so common, may occur in older children. Headache and extreme nervous irritability are common symptoms, and in older children neuralgic pain is a very common occurrence. Almost any nerve in the body may be affected, but the supraorbital is most commonly so. Severe intermittent supra- or infraorbital neuralgia, persisting after the acute symptoms have subsided, is strongly suggestive of sinus infection.

*Acute gastroenteritis* is very frequently caused by influenza; this manifestation is usually spoken of as intestinal grippe. It commonly follows, but it may occur quite independently of the catarrhal symptoms on the part of the respiratory tract. It is much more frequent in young than in older children, because at this age the bronchial mucus carrying infection is swallowed and the intestinal mucous membrane is perhaps less resistant. With the onset of this condition there may be nausea, vomiting, increase of fever and a sharp diarrhea. The discharges are putrid and contain large quantities of mucus which may be tinged with blood. The symptoms of an ordinary acute enterocolitis may follow, last for weeks, and place the patient's life in jeopardy; especially is this true in infancy. These cases occurring in older children may resemble typhoid fever.

An *erythematous rash* is frequently present during the acute stages of influenza. It may be very slight or it may be very marked, covering almost the entire body, presenting an exanthem very like that of scarlet fever; it commonly disappears within twenty-four or thirty-six hours. Other skin eruptions may appear, such as urticaria and a roseola somewhat similar to the rash of measles; these eruptions are all evanescent, and therefore do not commonly embarrass the diagnosis for more than twelve or twenty-four hours.

*Blood.*—Lord and other observers have found a slight leukocytosis in this disease, which becomes more marked when the influenza is complicated by septic processes, or when a more or less latent glandular tuberculosis has been rendered active by an attack of influenza.

The clinical picture of influenza above given may be greatly varied by the absence of certain symptom groups. In some instances the catarrhal symptoms on the part of the nose, throat and upper air passages may be very marked and very persistent, with slight fever and no nervous or other constitutional symptoms. In other cases the catarrhal symptoms of the

respiratory tract may be entirely absent, the fever and nervous symptoms predominating and producing a clinical picture quite unlike that of ordinary influenza. In other instances, especially in young children, gastro-enteric infection followed by an enterocolitis may occur without preliminary catarrhal symptoms on the part of the respiratory tract. That is to say, this disease may present itself in three well-marked symptom groups: the first and most characteristic is produced by catarrh of the respiratory passages; the second by the systemic intoxication; the third by gastro-enteric infection. The clinical picture may present a combination of these three groups, any one of which may predominate, or may be absent.

In infants and very young children the clinical syndrome of acute influenza may present certain peculiarities. The general infection is more severe and more sudden in its onset. Vomiting, convulsions, lack of appetite, apathy, stupor, opisthotonos and other symptoms closely simulating a beginning meningitis may occur. The fever is higher and rises more suddenly. Gastrointestinal infection with resulting catarrh is much more frequent. The erythematous exanthem is more frequently seen. The catarrhal symptoms appear later. The coryza is not generally so marked. The bronchitis which occurs late is much more serious than in older children, and pneumonia is more common.

Chronic Influenza.—The course of an uncomplicated influenza varies from three days to two weeks, and within this time the patient should and commonly does entirely recover. A few cases, however, by reason of the fact that they have influenza bacilli concealed in their tonsils, adenoids or some of the accessory sinuses of the nasopharynx, suffer from repeated mild relapses of attacks, the disease in this way becoming chronic. These cases frequently have enlarged and diseased adenoids or tonsils; they suffer from a low fever which may, for a few days at a time, reach normal or even fall below normal, to be followed again by a slight rise of temperature, rarely above 102°F. These acute exacerbations of fever may be associated with headache and general discomfort and in older children periodic neuralgias may occur. The patient fails to regain his appetite, is weak, anemic, and loses in weight and strength. The catarrhal symptoms on the part of the respiratory mucous membrane are more or less prominent; a spasmodic cough, resembling whooping-cough, may continue for weeks, but differs from the whooping-cough paroxysm in that it is less violent, is not aggravated at night and is not usually accompanied by the whoop or followed by vomiting. The rhinitis, while not very acute, is commonly present to a greater or less degree.

**Immunity.**—One attack does not confer immunity for any great period of time, but it does offer a degree of temporary protection. It is a matter of clinical record that influenza has appeared in a milder form since the great epidemics of 1889 and 1891; this is perhaps due to the fact that a great percentage of the population have, from previous attacks, acquired a certain degree of immunity.

**Complications.**—Otitis media is such a common complication that the

ear drums should be examined in every case. In this condition the influenza bacilli are associated with septic organisms and may produce a mastoiditis. The frontal and ethmoidal sinuses may be affected, especially in older children. Albuminuria occurs very commonly in children suffering from influenza. Acute hemorrhagic nephritis may develop very suddenly during the height of the disease; these cases not infrequently have a fatal termination. Post-grippe nephritis is less violent and runs a much more benign course than the hemorrhagic form. I believe that a large percentage of the cases of so-called idiopathic nephritis as well as those supposed to be produced by exposure to "cold," are cases of influenzal nephritis, having their origin in a recent attack of this disease; I also believe that influenza is one of the most common causes of chronic nephritis, and I feel quite sure that our text books and medical literature have not given to this subject the prominence it deserves. Tuberculosis is one of the most serious and common complications; an attack of influenza may aggravate an existing pulmonary or lymph-node tuberculosis. A prolonged bronchopneumonia with migrating areas of consolidation may be produced by the influenza bacillus. Conjunctivitis and other inflammatory conditions of the eye may occur. The heart may be overwhelmed by the toxemia, much as it is in diphtheria, and a myocardial weakness may persist for months after the acute symptoms of the disease have disappeared. In rare instances a splanchnic paralysis may occur.

**Diagnosis.**—It is practically impossible to differentiate mild cases of influenza from other catarrhal conditions of the respiratory passages. The pneumococcus, the micrococcus catarrhalis, and other microorganisms produce similar conditions of the nose, throat and bronchi, which can only be differentiated by an early bacteriological examination. In private practice this is rarely resorted to, as the differential diagnosis of these conditions from a clinical standpoint is not very important, since we have no specific treatment, and all are treated alike in a purely symptomatic way. From tuberculosis, influenza can be differentiated by the absence of the tuberculin skin reaction and by the failure to find tubercle bacilli in the sputum; from meningitis, by an examination of the cerebrospinal fluid and by the subsequent history of the case; from typhoid fever, by the absence of rose spots, the Widal reaction, and other symptoms of typhoid.

**Prognosis.**—The prognosis of uncomplicated influenza is almost always good. In rare instances young children are overwhelmed by the toxemia and death may result from cardiac paralysis, cerebral congestion, or intestinal toxemia. Apart from this the danger lies in its many complications, such as bronchopneumonia, acute Bright's disease, and mastoiditis, which may result fatally.

**Prophylaxis.**—Patients suffering from acute influenza should be isolated from other members of the household. This is especially important during the early acute catarrhal stage, as this is the period of greatest infection. The very young, the old, and individuals suffering from tuberculosis and other chronic diseases should be protected from this contagion,

since among this class of patients the disease is unusually severe and its complications especially dangerous. House disinfection is a most important prophylactic measure; in homes that are infected with this contagion the disease may continue to recur among the children of the family from time to time throughout the winter and spring months; formaldehyde disinfection may prevent these reinfections. Catarrhal discharges from the respiratory passages should be destroyed. Individual prophylaxis is also of importance. Much can be done by having the well children of the family spray or douche their noses and throats once a day with a mild alkaline antiseptic. They should also spend as much time as possible out of doors, and their physical condition should be looked to, if necessary, by the administration of cod-liver oil, iron and other tonics. This is especially important in families having a tuberculous family history.

**Treatment.**—The patient should be confined to bed during the acute stage of the disease. Rest in bed is a most important curative measure. The diet should be simple, suited to the age of the child, and especially selected with reference to throwing little work upon the excretory organs. Milk, cereals, bread and in older children fruit juices should be recommended. Albuminous foods, such as meats and eggs, are to be avoided during the acute stage, and but sparingly allowed during early convalescence. The patient should be induced to drink as much water as possible, as this helps to modify the febrile symptoms and to promote the excretion of poisons. A lukewarm tub bath once or twice a day is not only grateful, but is a valuable therapeutic measure, as it quiets the nervous symptoms and promotes elimination. The medical treatment should begin with a dose of calomel. This is to be followed by a mild saline cathartic such as phosphate of soda or Rochelle salts, and throughout the course of the disease mild cathartic medication may be necessary. Quinin is the most valuable remedy in the treatment of influenza. In children under five years of age from 1 to 4 grains of euquinin may be given every three or four hours. Quinin in this form can usually be administered without producing gastric disturbance; if discomfort follows its use, it should be discontinued. In children over five years of age the bisulphate or some of the other preparations of quinin may be given in pill or capsule, or combined with chocolate or licorice as recommended in the chapter on Malaria. Benzoate of soda is a remedy of value in the routine treatment of influenza. It should be given every four hours in doses of from 1 to 5 grains, according to the age of the child. It may be advantageously combined with tincture of belladonna. Phenacetin is a drug almost universally used and almost universally abused in the treatment of influenza. It is, however, of value when judiciously given. Of all the coal-tar products it is perhaps the least objectionable. It may be given to modify the headache, fever, and distressing symptoms at the very onset of the disease, but should not be continued longer than is absolutely necessary. It adds very materially to the comfort of the patient during the first two or three days, but exercises no curative influence on the disease, and if prolonged it may increase

prostration and cardiac weakness. For a child one year of age, 1 grain may be given every three or four hours, increasing the dose one-half grain for every year of life until the maximum dose of 3 grains is reached. Aspirin is a remedy of perhaps equal value with phenacetin and may be used in the same dosage. Salol is especially to be recommended for infants and young children and should be substituted for the phenacetin and aspirin in those cases where the acute discomfort is not great enough to demand the use of these drugs; it may be given in twice the dosage recommended for phenacetin.

Splanchnic paralysis and cardiac weakness occurring in this disease are to be treated as recommended in the chapter on Diphtheria.

The *coryza* and *pharyngitis* may be treated by local applications of mild antiseptics. In infants and young children 10 minims of the oil of eucalyptus may be combined with 1 ounce of liquid albolin, and this may be dropped into the nose with a medicine dropper at three or four-hour intervals. For older children 2 grains of menthol may be added to this prescription and this applied to the nose and throat with an atomizer. Weak alkaline antiseptic sprays may also be used to cleanse and disinfect the mucous membranes of the throat and nasopharynx; these may precede by one hour the albolin spray above mentioned. When the acute symptoms have subsided the course of the coryza and pharyngitis can be greatly shortened by the systematic use twice a day of these local applications.

The *cough* which accompanies the bronchitis and laryngitis of this disease requires treatment. For this purpose the bromid of soda and tincture of belladonna, put up with glycerin and some palatable elixir, may be used. For a child three years of age 4 grains of bromid of potash and 1 minim of tincture of belladonna may be given at four-hour intervals. Where the cough is very irritable and harassing heroin or codein may be combined with this prescription. Heroin hydrochlorate (1/150 grain) and codein sulphate (1/40 grain) may be given to a child five years of age. It is advisable, however, to avoid opium and all its derivatives, when possible, and they should rarely be given to children under two years of age. Acute gastroenteric infection and resulting enteritis are to be treated as directed in the chapters on these conditions.

In influenza the physician should bear in mind that we have no specific treatment, and that the condition is a self-limited one, which has a tendency to run a benign course even if no medication is used. The medical treatment, therefore, should be as simple as possible, and only such drugs used as are especially demanded by the symptoms present. Chronic influenza yields most readily to climatic treatment. These cases should be sent to a warm and equable climate where the patient may live out of doors.

**After-treatment.**—It is most important that the physician should be thoroughly awake to the fact that an active bronchial lymph-node tuberculosis is not uncommonly lighted up by an attack of influenza; in the after-treatment of these cases a warm equable climate, cod-liver oil and iron are most important. Many cases of influenza, in which there is no trace

of tuberculosis, are left in an anemic and weak condition; they are also benefited by the cod-liver oil and iron treatment. In children who have suffered from prolonged attacks of influenza, characterized especially by catarrhal conditions of the nasopharynx, a careful examination of the tonsils and adenoids should be made, and if these tissues be diseased or enlarged they should be removed; this is a most important curative measure. In many instances, in children suffering from recurring attacks of catarrhal influenza, I have, in the interval between these acute attacks, when the catarrhal symptoms were more or less in abeyance, had the tonsils and adenoids removed, with the result that a troublesome cough which had persisted for months would disappear and other catarrhal symptoms gradually subside.

## CHAPTER XXXIX

### SCARLET-FEVER

### (*Scarlatina*)

Scarlatina is an acute infectious and very contagious disease, characterized by fever, sore throat and a punctate scarlet rash which may cover the entire body and which is followed by widespread desquamation of the superficial epithelial layers of the skin.

**Etiology.**—Predisposing Causes.—Scarlet fever is a disease of childhood; it may, however, occur at any period of life. The young adult gradually acquires more or less immunity, so that in middle life the disease is comparatively rare, and much less severe, often manifesting itself as a mild attack of scarlatinal angina. It is also very rare during the first year of life. The comparative immunity which is enjoyed by the young infant is probably due to immune bodies derived from placental blood and is kept up to a degree by the breast milk. McCollom says that young infants are more susceptible than children of any other age; Heubner, on the other hand, never saw a case under six months of age. The explanation of these widely differing opinions lies in the probability that it is the nursing infant only that enjoys this comparative immunity, and that bottle-fed infants, however young they may be, are perhaps as susceptible to this disease as older children. After the first year of life scarlet fever increases in frequency up to the sixth year; the largest number of cases occur during the sixth and seventh years; thereafter the disease diminishes until, at sixteen, it is rather infrequent and later becomes comparatively rare. The susceptibility of the individual child is an important and unexplained factor; only about one-half of the children at the most susceptible age contract the disease even when they are brought into very close contact with the contagion. Scarlet fever is more prevalent during the winter than the summer months. The influence of cold weather in spreading this disease may be explained by the fact that it is during the winter months

that children are housed together in school-rooms and in their homes, thus producing conditions which favor the spread of contagious diseases, while during the summer months they live an outdoor life and are altogether under better hygienic surroundings. Minor, in his summary of the geographical disposition of scarlet fever, apparently demonstrates that the disease thrives best in temperate climates. He found that in the Western Hemisphere scarlet fever occurred between the 10th and 30th degrees, N. latitude, and that above and below this were zones of comparative immunity, in which the disease did not thrive for any length of time, even if imported.

Exciting Causes.—A microorganism, as yet undiscovered, is the cause of scarlet fever. The lower animals so far as we know are not susceptible, and we have no evidence that the scarlet fever germ can multiply outside the human organism. There is evidence, however, that it may live and be transported in milk, the milk acting as a carrier rather than as a culture medium. The specific cause of scarlet fever is very tenacious of life, and may live for a long time under very adverse circumstances. It may cling to bedding, carpeting, hangings, clothing, linen, to the wall-paper and to apparently everything with which it comes in contact. It is especially difficult to eradicate from infected rooms; many instances are on record where such rooms have been cleaned and apparently disinfected, and yet months later the disease has been contracted by children who have moved into them. The contagion of this disease lies especially in the muco-pus from the nose, throat, ears and in the scales of epithelium cast off by the dermatitis. The air immediately surrounding the patient is apparently not contaminated for more than four or five feet, and this contamination probably results largely from the spray of mucus that is coughed into the surrounding air. Dried mucus and fine epithelial scales carrying contagion may be swept or otherwise thrown into the air, and perhaps be wafted for slight distances. Human intercourse, which brings the well in contact with the sick, is the great cause of the spread of this disease. In older children it frequently manifests itself as a scarlatinal angina; such children may never be seen by a doctor, and may continue to go to school, or to children's parties, or to mingle freely with the other children of the household, spreading the disease in their wake; this is perhaps the most common way in which the disease is disseminated. Children that are supposed to be convalescent, but who still have otitis media, rhinitis, or slight desquamation, are very frequently turned loose upon the community while they are still capable of spreading the infection. The poison may also be carried by letters, and by cats and dogs which pass from the sick to the well. Epidemics are also reported in which contaminated milk was supposed to be the carrier. Attendants and nurses may spread the disease in street-cars and homes to which they go after a night or day of nursing. The doctor, *if he takes proper precautions,* should not be a source of danger. After many years of experience in hospital and private practice, I have never had a case in which there was the least suspicion that I had

been the carrier of the contagion. The infection commonly enters through the mouth or nose and affects primarily the mucous membrane of the nasopharynx. It may, however, enter surgical or other wounds, or it may, by the hand of the obstetrician, be carried into the vagina. These latter methods of infection are now very rare.

**Period of Contagion.**—Scarlet fever is contagious from the appearance of the first catarrhal symptoms in the throat. The most contagious period is during the first week, when the fever and throat symptoms are severe. With the subsidence of these symptoms the disease is less contagious, but with the beginning of desquamation it again becomes more contagious, and contagion probably exists as long as desquamation lasts, or as long as there is a mucopurulent discharge from the nose, ears, and throat. In all well-marked cases it is safe and proper to assume that the contagion lasts for at least six weeks, and during this time there should be a rigid quarantine. The proper care of the skin, the disinfection of the throat and general hygienic measures for destroying the contagion and preventing the contamination of the surroundings will diminish the period of contagiousness.

**Pathology.**—The specific organism of scarlet fever is unknown, but streptococci play a most important rôle in its pathology. These cocci, which are almost always found in the throat and often in the blood, have been believed by many writers to be the actual cause of this disease. Klein and Gordon described a streptococcus scarlatina and Kurth a streptococcus conglomeratus, these organisms differing slightly from the streptococcus pyogenes which has been so generally associated with the pathology of scarlet fever. The streptococci which have been found in the throat and blood of scarlet fever patients have no well-defined characteristics which differentiate them from other streptococci. The streptococcus pyogenes and the staphylococcus aureus and albus are more or less definitely associated with the destructive processes which accompany and follow scarlet fever. Hektoen found that the degree of streptococcemia in scarlet fever was closely related to the severity of the disease. In mild cases few streptococci were found in the blood; in severe, and especially in complicated, cases they were found in larger numbers. They may, however, be absent from the blood in even fatal cases. Streptococci are also found in the urine and in the discharges from the nose, throat, and ears. Mallory describes certain protozoön-like bodies found in the skin of scarlet fever cases which are of interest, but their pathological and etiological importance has not yet been determined. Vipond isolated a bacillus with which he produced in monkeys a scarlet rash and fever.

The lesions in scarlet fever are not characteristic; well-defined, acute dermatitis, ending in desquamation, is the most distinctive. The angina, which produces a marked congestion and inflammation of the tonsils, pharynx and soft palate, with a grayish-white exudation due to the action of cocci, is also commonly present. The other lesions on the part of the lungs, kidneys, joints, lymphatic glands, and cellular tissue are complica-

tions due rather to the action of cocci than to the specific organism which produces scarlet fever.

**Period of Incubation.**—The period of incubation has perhaps a wider range than in any of the other acute infections. According to McCollom, it varies from four to twenty days, the average period being ten to fourteen days. Most other writers name a shorter period of incubation, the average being six or seven days. A number of well-authenticated instances are on record where it has developed within twenty-four hours after exposure, and the evidence also seems to be conclusive that the incubation stage may be prolonged for fifteen or twenty days.

**Symptomatology.**—Scarlet fever is a disease that presents the widest variations in its symptomatology, from a mild angina which may not be recognized to foudroyant cases where the toxemia is so intense that the patient's life is destroyed within twenty-four hours. The following description represents a moderately severe case of typical scarlet fever; the variations from this type will be considered later.

Onset.—A feeling of malaise, covering a period of one or two days, may precede the more characteristic symptoms, but in the great majority of cases the onset of the disease is marked by vomiting, headache, fever, and sore throat. This symptom group, which is more or less suggestive of scarlet fever, may be accompanied by a chill in older children or convulsions in young children. Within the first twenty-four hours a scarlet *rash* commonly appears on the chest and neck, and gradually extends over the body. During the second week desquamation, the most characteristic of all the symptoms, makes its appearance. None of the above symptoms, however, are absolutely characteristic, any of them may be absent, but in the great majority of cases the syndrome of scarlet fever made by the above symptoms is sufficiently distinct to make a diagnosis. The severity of the disease may be predicted in a measure by the suddenness and violence of its onset.

*Vomiting* occurs in about 70 to 80 per cent. of the cases and commonly marks the onset of the disease. It may be repeated a number of times and then subside. Prolonged and continuous vomiting is not characteristic. When vomiting occurs late, after the other acute symptoms have subsided, it is a more serious symptom and may mean a beginning uremia. In young children a diarrhea may accompany the vomiting, but this symptom rarely persists longer than two days.

*Fever.*—A rise in temperature immediately follows the vomiting. The fever commonly reaches its height by the end of the second day, but the maximum temperature may, in some cases, be found at the end of the first twenty-four hours; a temperature of 102° F. indicates a mild infection, and 105° F. or over, a severe one. Following the rapid rise in temperature of the first two days, the fever usually begins to subside, getting lower day by day until by the end of the week it may reach normal. It may, even in uncomplicated cases, last from twelve to fourteen days. There is nothing characteristic in the temperature curve of scarlet fever, and its value

as a diagnostic sign depends largely upon its association with the other symptoms. A rise in the temperature, after it has been slowly falling for a number of days or after it has become normal, indicates some complication, such as adenitis or otitis.

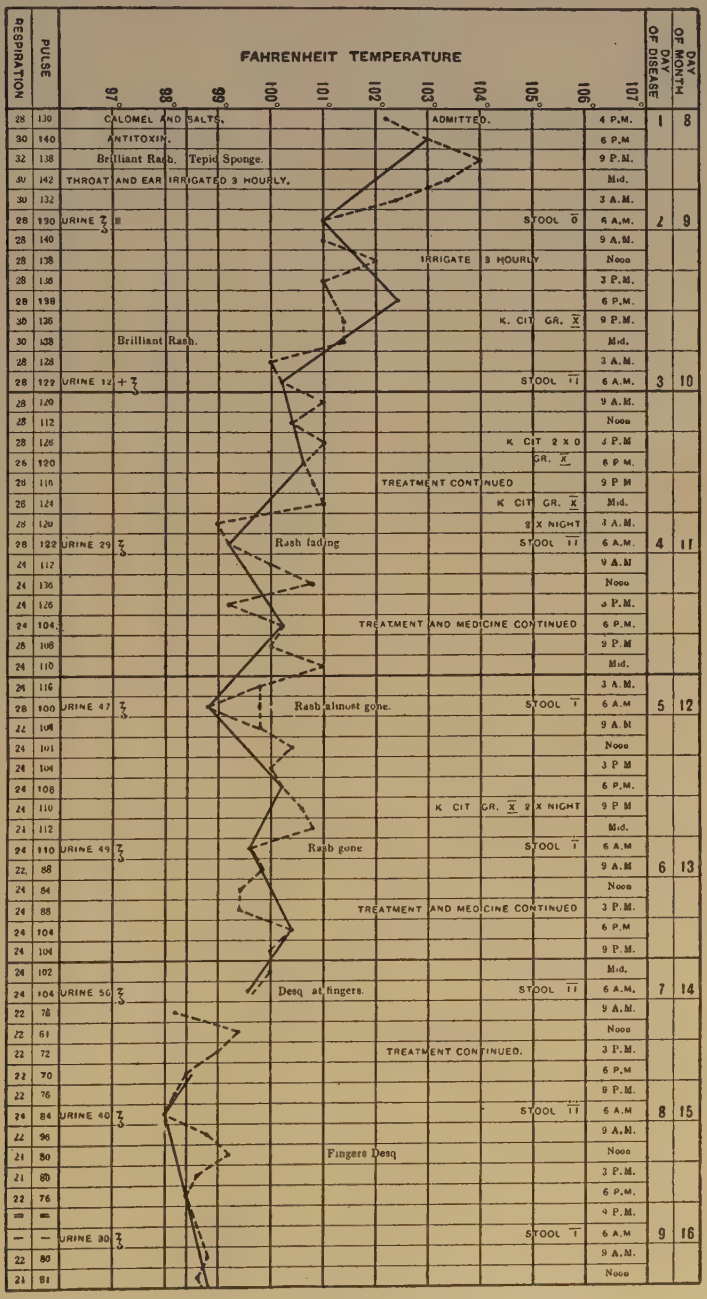



FIG. 52.—SCARLET FEVER, MILD; CHILD TWELVE YEARS OF AGE.

The *pulse* rate in scarlet fever is usually high; in even mild, uncomplicated cases with a temperature below 103°F. the pulse may run from

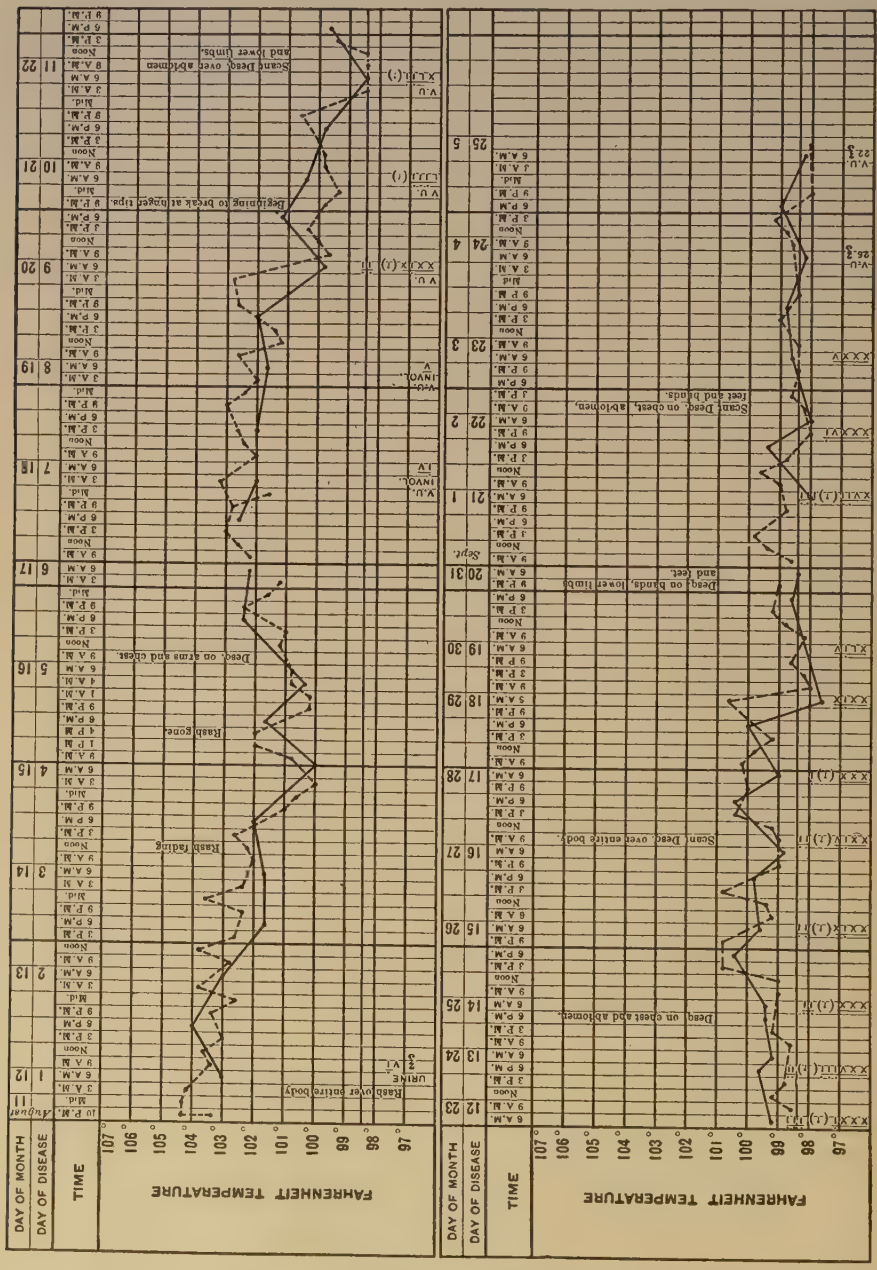



Fig. 53.—Scarlet Fever, Severe; Child Six Years of Age.

140 to 160; in the more severe cases it may reach 170 or 200 per minute, but this high pulse rate, out of proportion to the temperature, does not

necessarily mean an unfavorable prognosis; it is, however, a symptom of diagnostic value.

*Sore Throat.*—The sore throat which is almost always present is, when taken in connection with the other symptoms, of great value in diagnosis. It manifests itself in four types: (*a*) Simple pharyngitis with swelling of the pillars of the fauces and tonsils; (*b*) more intense swelling and infiltration of these tissues with grayish white deposits in and around the tonsillar crypts; (*c*) diphtheroid angina with intense infiltration of pharyngeal structures, associated with adenitis and cellulitis of the neck; (*d*) true diphtheria. In the early examination of the mouth in suspected scarlet fever cases, one may sometimes make out that the roof of the mouth is not only red and congested, but that it has also fine points of deeper redness scattered over it, which give it a punctate appearance. This enanthem when present is a valuable diagnostic sign.

Other General Symptoms.—*Cervical adenitis* of the glands at the angle of the jaw occurs in the majority of cases. When the throat inflammation is severe, ulcerative and necrotic, the cervical lymph nodes are more seriously involved. These glands may enlarge, forming tumor masses bound together by an inflammation of the cellular tissue which may result in a more or less board-like tumefaction. This condition is an ominous one; it may result in a dry necrosis, causing great destruction to the tissues of the neck and a subsequent general septicopyemia. This severe form of adenitis is to be considered rather as a complication than as a symptom of the disease, but even the milder cases may result in a suppurating adenitis, especially in tuberculous children. This process occurs more commonly in children suffering from latent tuberculosis.

*The Tongue.*—The appearance of the tongue is of more diagnostic value in scarlet fever than in any other general disease. In the beginning it is covered with a white coat through which small red papillæ are seen to protrude; after two or three days the white coating disappears, leaving it red, but the papillæ still stand out prominently as little dots of redder hue; these are to be seen along the edges and especially on the tip of the tongue, and give to it a strawberry appearance, hence the term "strawberry tongue," first described by Flint.

*Eruption.*—The rash of scarlet fever generally makes its appearance on the neck or chest within the first twenty-four or thirty-six hours; it may, however, be delayed until the third or fourth day, and in rare instances even as late as the sixth day, and is usually accompanied by more or less itching. The eruption, within two days after its appearance, spreads to all parts of the body. In the beginning it has a punctate appearance produced by small red points closely approximated and scattered in patches over the skin, which coalesce, and in the course of twenty-four hours the punctate appearance may be lost in a uniform scarlet eruption which disappears on pressure. The rash takes on a deeper scarlet hue and reaches its maximum of redness in two days; it then gradually fades, entirely disappearing in from four to seven days. The face may remain free or show

only slight signs of the eruption, but even when the skin of the face is covered with the scarlet rash there is a peculiar pallor about the mouth, and herpes may also be present. The rash is more intense in those regions of the body where the folds of the skin are brought into juxtaposition, such as the axilla, the inguinal region, the under-surface of the elbow and knee joints, the folds of the buttocks, and approximated surfaces of the thighs. The rash of scarlet fever varies greatly in different cases; it may not be present at all, it may last but a few hours, it may appear in but a few punctate patches on the parts of the body where the skin is in juxtaposition, or it may present the typical appearance just described and be followed by miliaria or urticaria. In malignant cases it may be hemorrhagic.

*Desquamation,* which is perhaps the most characteristic symptom of scarlet fever, commences about the seventh day; in severe cases it may begin earlier, in mild cases later. It continues from three to six weeks, or in severe cases longer. The character and extent of the desquamation depend largely upon the severity of the dermatitis; if this be severe and marked by a uniformly brilliant scarlet eruption, the desquamation that follows is more extensive and the epithelium is peeled off in larger flakes. In the milder cases it may be almost or quite absent; in such instances it should be sought for especially in the axillary and inguinal regions, and under the finger nails. Desquamation begins, as a rule, on the neck, chest, and fingers and spreads to other portions of the body, the skin of the feet being the last to peel. It may be fine and scaly, like the furfuraceous desquamation of measles; it is, however, more commonly flaky or lamellar in character. The older the child, and the more hardened and tougher the skin, the more marked the desquamation. On the hands and feet the epidermis may be peeled off in large pieces; entire casts of the fingers and hands have been removed in some instances. Secondary desquamation may occur, especially in the more severe cases.

Urine.—The urine should be examined frequently from the onset of the disease until desquamation has ceased. A trace of albumin is present in from 10 to 15 per cent. of the cases; this slight albuminuria, even when accompanied by a few hyalin casts, is not an ominous symptom. It may be a simple febrile or toxic albuminuria, more commonly present in this disease than in other fevers, because the skin is largely put out of action and the kidneys are therefore called upon to do extra work. The presence of albumin, however, associated with granular and epithelial casts, is a much more serious matter, and indicates the onset of acute nephritis. The kidney complications of scarlet fever are commonly post-scarlatinal, occurring after the acute symptoms have subsided. This subject is considered in the chapter on Acute Nephritis.

Blood.—There is a slight secondary anemia with a moderate reduction of both hemoglobin and red blood corpuscles. Between the second and eighth day there is a leukocytosis of eighteen to forty thousand; the polymorphonuclear leukocytes are relatively and absolutely increased early in

the disease, and rapidly diminish as the fever and toxemia subside. The mononuclears are relatively and absolutely increased in the later stages. The eosinophiles are increased as the toxemia subsides.

**Recurrence and Relapse.**—One attack of scarlet fever confers permanent immunity. This is a rule which has but few exceptions, although very rarely second attacks may occur. A relapse, while also rare, is more common than a recurrence from reinfection; it may occur during the third or fourth week and produce the symptom group in a milder form. A return of the eruption within two or three days after its disappearance is not to be confounded with a true relapse, since its return at this time is usually of little moment, and it disappears shortly without being accompanied by other untoward symptoms.

**Irregular Clinical Types.**—From the ordinary type above described there are a number of important clinical variations.

Mild Type.—Scarlet fever may manifest itself in a form so mild that the character of the disease may not be suspected and the infected child may unintentionally spread the disease broadcast. Vomiting may or may not be present, there is a slight elevation of temperature, which soon falls to normal, and the patient is thought to have an ordinary tonsillitis or pharyngitis. The true nature of the infection may be determined by previous or subsequent cases of scarlet fever in the same family or by the appearance of a slight eruption, followed by furfuraceous desquamation in the axilla or groins. The enanthem or fine punctate eruption on the roof of the mouth may be present, and should always be looked for in every case of sore throat or scarlatinaform eruption.

Malignant or Fulminating Type.—Cases of this character are very uncommon. Within the course of a few hours the child may be so overwhelmed with the poison that the hopelessness of the case is apparent. In the most severe cases death may occur within two days; as a rule, however, the disease lasts from three to six days. The onset is marked by severe nervous symptoms which may suggest meningeal involvement; convulsions, delirium and coma may follow each other in quick succession. The child tosses about in bed and cannot be quieted; the fever from the onset is high, the pulse rapid, and death commonly results either from acute cardiac dilatation or from slow cardiac failure. If the patient lives long enough a severe sore throat and scarlet eruption appear; in some instances the rash is hemorrhagic. In the most violent of these cases the diagnosis is difficult and at times impossible, as the patient does not live long enough to develop typical symptoms. Between the mild and malignant types we may have every grade of severity. In the same epidemic we may see both mild and malignant cases.

Septic Type.—Accurately speaking, this is a complication rather than a type of scarlet fever, but as previously noted, the rôle played by septic organisms in producing the symptom-complex of scarlet fever is very great, and in a large percentage of the cases the septicemia or septicopyemia presents such a distinct symptom-complex that it has come to be considered

as a type of this disease. The onset is that of severe scarlet fever, but as the fever begins to subside a slight or sudden rise in the temperature occurs. This secondary rise is a most significant symptom, and is usually accompanied by an enlargement of the cervical lymphatics. Upon these two symptoms alone one is justified in assuming that the time has come to use our most energetic therapeutic measures for controlling septicemia. If the condition is not controlled, the temperature continues high with marked variations and may run for many weeks. At times it is not unlike the temperature of typhoid fever or of general miliary tuberculosis. The throat symptoms and all other symptoms of scarlet fever may gradually disappear, leaving the symptom-complex of a septicopyemia. Suppuration of the lymphatic glands of the neck may occur and a septic pericarditis, arthritis, pleuritis or bronchopneumonia may develop.

**Complications.**—*Otitis media* is a very common complication. It occurs in from 15 to 20 per cent. of hospital cases, but is less in private practice; it is a sequel of the pharyngitis of this disease and is more common in tuberculous children. In the treatment of every case of scarlet fever, and especially in those with tuberculous family histories, the possibility of the development of otitis media should constantly be kept in mind and a secondary rise in temperature should always lead to a careful examination of the ear. In infants, apparently convalescent from scarlet fever, this complication may be suspected if the child suddenly becomes restless, sleepless and cries with pain.

*Ulcerative* and *gangrenous angina,* which may result in the destruction of the tissues of the throat, is much to be dreaded but is fortunately rare.

The *septic arthritis,* which may occur as a part of the symptom-complex of the septicopyemia of scarlet fever, is multiple, commonly involves the large joints and is sometimes associated with purpura. There is, however, another form of scarlatinal arthritis, producing an acute inflammation of the synovial membranes, not associated with pus formation. This condition is spoken of as scarlatinal rheumatism. It is a rare but very well defined symptom group; the wrist and finger joints are most commonly involved, but some of the large joints of the extremities may also be affected. It produces fever, with redness, tenderness and swelling of the joints, and it may produce pericarditis and true endocarditis, leaving the valves of the heart permanently injured. Both the arthritic and cardiac symptoms, however, are on the whole much less severe than they are in true articular rheumatism. Salicylic acid apparently relieves the pain and reduces the fever which occurs in this condition; whether or not it is true rheumatic fever, complicating scarlet fever, or a manifestation of the scarlatinal toxemia is not altogether clear. The latter hypothesis is more rational.

*Nephritis* is one of the most common and serious complications of scarlet fever. In the majority of instances it is a post-scarlatinal lesion and is, therefore, to be carefully looked for during and after the third week. It may be made manifest by a slight puffing of the eyelids, severe head-

ache, sudden rise of temperature, nausea or vomiting, but the diagnosis is made by the finding of albumin and casts in the urine, or possibly by the sudden development of convulsions and other uremic symptoms.

*Cardiac murmurs* are common during the height of the disease, but true endocarditis is rare. Myocardial degeneration of greater or less degree is very common.

*Nervous lesions* may occur. In severe cases meningitis, hemiplegia, chorea, and symmetrical gangrene are occasionally seen.

**Diagnosis.**—There is perhaps no disease in which it is more important to make an accurate diagnosis than in scarlet fever. The responsibility of the physician is here very great. On the one hand if he comes in contact with a mild case and fails to make the diagnosis great damage may be done by spreading the disease broadcast. On the other hand, if he comes in contact with one of the numerous rashes that so closely resemble scarlet fever, and condemns the patient as well as the entire household to six long weeks of rigid quarantine, he has thereby done great injustice and caused great inconvenience. Since in many instances it is absolutely impossible for the physician to make the diagnosis it is his duty to quarantine all suspicious cases until the question has been fully settled. In the ordinary case the diagnosis is easily made by the sudden onset of vomiting, sore throat and fever followed by the rash, the "strawberry tongue" and later the desquamation. Upon one point in the differential diagnosis I wish especially to insist, and that is upon the early appearance of the more or less typical enanthem. The roof of the mouth is red and congested, and over this red surface fine points of more scarlet hue are scattered in close juxtaposition. When this condition can be made out it is of great value as an early diagnostic symptom in distinguishing scarlet fever from measles and rubella.

The erythematous rash of influenza associated with fever, sore throat and gastric disturbance produces a clinical picture which can only be differentiated from scarlet fever by the development of further symptoms. Rashes resembling scarlet fever may also result from digestive disturbances, sepsis and from drugs such as antipyrin, quinin and atropin. Acute exfoliating dermatitis and the serum rashes following the use of diphtheria antitoxin are often difficult of differentiation from scarlet fever.

**Prognosis.**—Age is one of the most important determining factors in the prognosis of scarlet fever. The younger the patient the more fatal the disease. From a study of 5,000 cases of scarlet fever, treated at the South Department of the Boston City Hospital, McCollom found the death rate to be over 33 per cent. in children under one year of age. This mortality rapidly decreased so that between the sixth and seventh year it was about 7 per cent. From this time on there was a very gradual decrease in the death rate. The accompanying chart graphically illustrates the increasing powers of resistance which age gives to this disease.

The character of the epidemic is also important in determining the death rate. Some epidemics are characterized by unusually severe cases

with great mortality. Others are mild and the mortality is correspondingly low. The mortality, especially during the first two or three years of life, is much lower in private than it is in hospital practice. This is not only due to the early medical attention which private cases receive, but also to the fact that these children are much better nourished and have, on the whole, greater powers of resistance than the malnourished weaklings that find their way to public hospitals. The general mortality of scarlet fever at all ages is variously estimated by different writers; it is on the

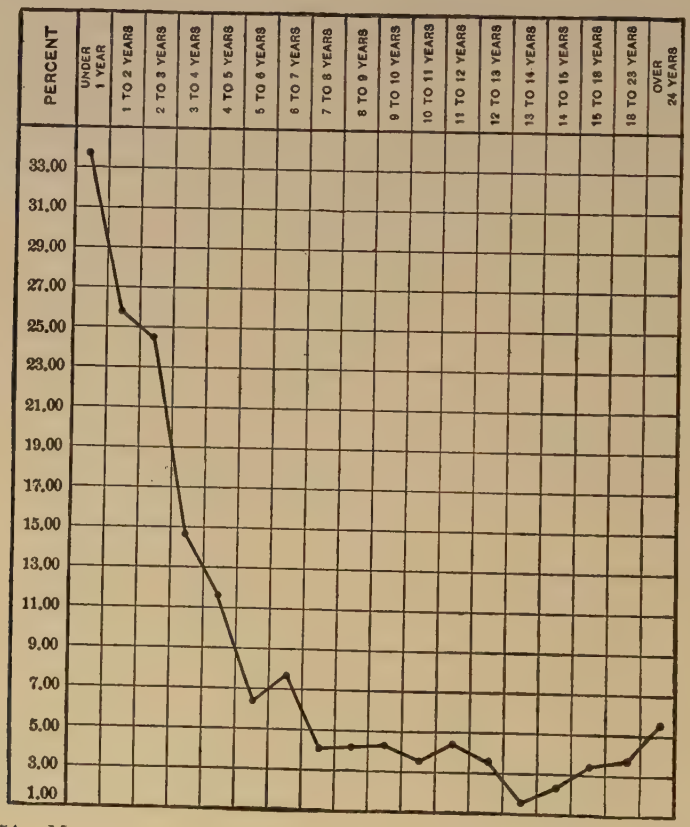



Fig. 54.—Mortality by Age in 5,000 Cases of Scarlet Fever. (McCollom.)

average about 12 per cent. In the individual child, apart from its age and the severity of the prevailing epidemic, the severity of the onset is the most important prognostic factor. High temperature, convulsions, profound nervous symptoms, and a bad sore throat with extensive involvement of the cervical lymph nodes indicate that the child's life is in peril. Dangers may beset the child from the onset of the disease until the completion of its convalescence. In the beginning the danger is that it may be overwhelmed by the violence of the toxemia. Toward the end of the

first week, violent throat symptoms with a beginning septicemia may foretell an unfavorable termination, and throughout its convalescence, nephritis, bronchopneumonia and a general septicopyemia may place the child's life in peril. With all of these unforeseen dangers the prognosis in any individual case is uncertain, even though the present conditions are favorable.

**Prophylaxis.**—The prophylactic treatment of scarlet fever is perhaps more important than that of any other disease. The most important object to be obtained is the absolute isolation of the sick child from everyone except the necessary attendants. This is a matter not only of the greatest difficulty, but, in the majority of instances, it is absolutely impossible. Among the children of the poor, the patient should be removed to a contagious-disease hospital. Among the rich, the problem of isolation is not so difficult. Among the middle class, the question of expense, which absolute isolation entails, is a very serious hindrance to the proper prophylactic treatment of scarlet fever. There are, however, certain recognized principles in the home treatment which the physician should endeavor to follow, conforming as closly to the ideal conditions as circumstances will allow. The following details apply not only to scarlet fever but to diphtheria and smallpox as well.

As soon as scarlet fever is suspected the two most available rooms in the house, for isolation purposes, should be selected; one for the patient and one for the nurse off duty. All rugs, carpets, hangings, and unnecessary articles of furniture and clothing should be removed, and the rooms furnished with iron beds and with such chairs and tables as can be easily cleaned. A day and night nurse should be installed, and they should have entire charge of the rooms and the patient. The fetching and carrying of food, clothing and other things to and from the room should be done with such care that other members of the household will not be infected. The physician on entering the room should put on a fresh white gown and cap, and should remain in contact with the patient as short a time as possible, and on his exit should carefully cleanse and disinfect his hands, his stethoscope, or any other instrument he may have used. The nurse should be directed to destroy the excretions from the throat and nose, either by burning or by placing them in a solution of bichlorid of mercury. There is no danger that the poison of scarlet fever will permeate the air and contaminate halls and neighboring rooms; for this reason isolation, when intelligently carried out, will protect the other members of the household from the disease. When desquamation commences, inunctions with oil or ointments will not only be a comfort to the patient, but will prevent the scattering of the scales of skin throughout the room; the frequent use of inunctions is therefore a most important prophylactic measure. The isolation of the child should be rigidly kept up until desquamation and all discharges from the nose and throat cease; in the average case this covers a period of six weeks. When the quarantine is raised the patient's body should be thoroughly washed with soap and hot

water, and his hair and nails carefully cleansed before he is permitted to leave the room and mingle with the household.

The sick rooms should be fumigated with formaldehyde. McCollom says: "The simplest and also an effectual method of generating formaldehyde is that adopted by the Maine Board of Health, which consists of mixing potassium permanganate with a 40-per-cent. solution of formalin. The potassium permanganate should be the commercial and not the chemically pure. No special apparatus is required. An ordinary tin dish with flaring sides can be used. The quantity of permanganate for each pint of formalin is 6½ ounces. It is very important that the permanganate be put in the dish first, and then the formalin solution poured over it. It is taken for granted that the room has been tightly sealed. As soon as the formalin is placed in the receptacle a rapid exit must be made, because the gas is generated very quickly. The room should remain closed for twenty-four hours and then be thoroughly aired. The quantity of the 40-per-cent. solution of formalin and of the potassium permanganate to disinfect 500 cubic feet of space is 1 pint of the former and 6½ ounces of the latter." Following disinfection, the woodwork should be washed with soap and water and then rubbed down with a 1:1,000 solution of bichlorid of mercury. The wall paper should be cleaned or removed. All books and toys should be destroyed and the bed linen should be treated, as throughout the course of the disease, by placing it first in a bichlorid solution and then subjecting it to boiling. The mattress should be burned. Where this is not expedient, as in hospitals, it should be disinfected by steam. Following the cleansing of the room, it should a second time be fumigated with formaldehyde.

School inspection by health boards is one of the most valuable prophylactic measures. Where this is systematically carried out at least once a week, a large number of children will be found with throat and nose disease, or with other evidences of illness which justify their being sent home and kept under proper medical supervision until they have recovered. In our large cities, where school inspection is now being successfully carried out, there is little doubt but that many children with scarlatinal angina are prevented from mingling and spreading the disease among other children. Clinical reports now indicate that the use of streptococcus vaccines may be a valuable prophylactic measure.

**Treatment.**—There is no specific treatment for this disease, and the physician should ever keep in mind that it is self-limited and in the great majority of instances runs its course to a successful termination. These facts being understood, it is most important that medication should be given only when medicines are indicated, and that the symptomatic treatment should not be overdone.

The patient should be put to bed and kept there for a week or ten days after all acute symptoms have subsided; this materially diminishes the danger of renal and other complications. The sick room should be well ventilated, at a temperature between 65° and 70° F. The bed and body

linen should be frequently changed, and the bed covering should be comparatively light. It is most important that scarlet-fever cases should not be kept overwarm with bed clothing and by living in superheated rooms. During the first forty-eight hours the bowels should be moved with calomel, followed by some saline laxative, and thereafter kept open, if necessary, by the use of some mild cathartic, such as cascara.

Dietetic Treatment.—During the first days barley water and mutton broth may be given. With the subsidence of the vomiting and the quieting of the stomach the milk diet is begun. Milk in some form should be almost the exclusive diet for the next three weeks; one or two quarts, depending upon the age of the child, may be taken within twenty-four hours. Milk is of special value in that it serves nutritional purposes, is easy of digestion, acts as a diuretic and throws very little work upon the already overtaxed kidneys. It will sometimes tax the ingenuity of the physician to maintain the milk diet throughout this period. The term milk diet, as here used, includes all of the artificial milk foods, ice cream and buttermilk. The milk may be flavored with cocoa or vanilla, mixed with cereal decoctions or made into milk-soups. Certain drinks, such as lemonade and orangeade, never mixed with albumin water, may be given. During convalescence, after the third week, other foods may be used, such as bread, stewed fruits, baked apples, rice pudding, cereals, thick soups, and, later, potatoes, vegetables, fish and chicken may be added.

Hydrotherapy is a most important part of the treatment. In all cases, especially where the temperature runs high, baths are not only a valuable therapeutic measure but they add greatly to the comfort of the patient. In the milder cases sponge baths, with water at a temperature between 70° and 80° F., should be given twice a day. In severe cases tub baths at a temperature of 70° F. or moderately cold packs given every four or six hours are more effective. In connection with these baths, an ice-cap should be applied to the head, but this application should not be continuous, except in the malignant type of the disease where the fever is high and the nervous symptoms are profound. It should be remembered, in applying the various measures here recommended, that all children do not respond kindly to cold baths; this is especially true of infants. On the whole, it may be said that the older the child the more effective and the more satisfactory will be the cold-bath treatment. But in any given instance, if the bath produces unfavorable symptoms, such as great prostration, weak heart, cyanosis, and cold extremities, it is to be either discontinued or so modified as to get the good without producing the bad results. The ordinary coal-tar antipyretics should not be used for the control of the temperature or the nervous symptoms.

Stimulants.—In malignant cases stimulants are necessary to counteract the severe onslaught of the toxins on the nervous system and the heart. For this purpose whiskey, brandy, or champagne may be freely given during the early days of the treatment. After the poison has spent its force and the nervous system has commenced to recover from its effects alcohol is

no longer indicated and finds no place in the treatment of scarlet fever; its use after this period may be injurious to the kidney. Other stimulants, however, may be used in connection with the alcohol and may be continued throughout the course of the disease; the most valuable of these are strophanthus and digitalis. They are especially indicated in severe and prolonged cases of scarlet fever, with rapid and weak heart. Strophanthus is especially valuable in very young children and digitalis in older children, and both of these drugs should be combined with essence of pepsin or some other palatable vehicle which will protect the digestive organs of the child. Strychnin in 1/150 to 1/200-grain doses is another valuable stimulant, acting especially on the respiratory centers, and may be continued throughout the course of the disease. In extreme cases, where collapse threatens, salt solution and caffein sodium benzoate may be given in the same dose and manner as directed under Diphtheria.

Inunctions are a part of the routine treatment of scarlet fever. The child should be anointed twice a day with oil, cocoa-butter, lanolin, cold cream, or lard. They prevent the scales of skin from being scattered about and make the patient more comfortable by allaying the itching and irritation of the skin. Seibert recommends the use of a 10-per-cent. icthyol lanolin ointment. Other writers advise that boric acid and carbolic acid be combined with the ointments above named. The general consensus of opinion is that the value of this treatment depends upon the soothing rather than the antiseptic action of the ointment.

The systematic treatment of the nose and throat is important; for this purpose an atomizer may be used containing 1 ounce of liquid albolin, combined with 10 minims of oil of eucalyptus and 3 grains of menthol, or some alkaline antiseptic solution. When the angina is severe, mild saline antiseptic solutions may be injected through the nose, coming out through the pharynx, in the manner described in the chapter on Therapeutics of Infancy and Childhood, and the throat should be swabbed alternately every two or three hours with a 1/1,000 bichlorid of mercury solution and a 30 per cent. argyrol solution. The object of this treatment is to minimize the systemic intoxication, to modify the local inflammatory conditions, and to prevent internal ear complications. In severe gangrenous inflammations of the throat strong nitrate of silver solutions and even the thermocautery have been recommended. Where diphtheria complicates scarlet fever diphtheria antitoxin should be given.

With the onset of septicemic symptoms one should begin the use of antistreptococcic serum made from cocci taken from a case of scarlet fever; from 100 to 200 c. c. should be given every eight to twelve hours for three or four days; the size of the dose will depend upon the age of the patient. In my experience this serum, if given at the proper time, is a life-saving measure of great importance. Escherich and Moser, using Moser's serum, greatly reduced the mortality of scarlet fever in the Annakinderspital at Vienna. Fedinski and Nicoll have also used antistreptococcic serum with very favorable results. Collargolum is a remedy of great value in the

treatment of the septicopyemia of scarlet fever. It may be given in the form of rectal suppositories or perhaps better by inunctions with unguentum Credé. This latter preparation I have used very extensively for years, and I believe that when properly administered it is of great value in preventing the development of general septicemia. The technique for its use is as follows: The whole upper portion of the child's body is carefully cleansed with soap and water, and the skin is then made hot by the application of warm fomentations. One-half ounce of unguentum Credé is then slowly and carefully rubbed into the upper portion of the child's body over the neck, chest, and axillæ, being careful not to injure the inflamed lymph nodes. This should be done twice a day for three or four days. The object of this application is to rub the silver ointment through the skin into the lymphatics and not simply to make an application over the enlarged lymph nodes. In the child this ointment is very readily taken up by the lymphatics and acts as a very potent lymphatic antiseptic.

The treatment of ADENITIS, in addition to the measures above outlined, may demand the local application of ice; light ice-bags applied intermittently exercise a favorable influence, especially in the early stages of the inflammation. Forchheimer recommends that pressure be applied to these glands by the application of flexible collodium; 49 parts of collodium to 1 part of castor-oil. If suppuration occurs, poultices are not only grateful, but hasten the breaking down of the gland, which is then to be incised with proper surgical precautions.

OTITIS MEDIA.—Scarlet fever is one of the most common causes of deafness. In every case, especially if there be a tuberculous family history, the physician should always keep in mind the fact that otitis media may occur and may not only cause loss of hearing but may threaten the life of the child by producing a mastoiditis. The ear should, therefore, be frequently examined throughout the course of this disease, and symptoms pointing to this complication should be constantly watched for. An early incision of the drum membrane may check the inflammation and prevent the involvement of the mastoid. The ear should then be carefully washed out twice a day with a boric-acid solution and should be carefully dried and some powdered boric acid dropped into the meatus, which is then lightly plugged with a pencil of dry cotton; this treatment should be continued from day to day. If there be the slightest tenderness over the mastoid, an ice-bag should be applied to this region, and the application of leeches may also be of value. If, however, the mastoid tenderness does not subside, and the septic temperature continues, the radical mastoid operation should be performed.

SCARLATINAL RHEUMATISM should be treated in the same manner as acute rheumatic fever. The joints should be kept warm by wrapping them in flannels or cottons, and salicylates, such as aspirin, should be given to relieve the fever and pain. If the symptoms do not respond quickly to the salicylate treatment it is to be discontinued, as there is some difference

of opinion as to whether the continued use of salicylic acid in this disease may not increase the dangers of nephritis.

Nephritis.—In every case of scarlet fever the urine should be examined daily for the onset of this complication, and if nephritis develops, hot baths, saline cathartics, a milk diet and the other treatment outlined under Acute Nephritis are to be carefully carried out.

**Convalescence.**—Scarlet fever, especially in tuberculous children, leaves the patient more or less anemic, with enlarged lymphatic glands and possibly with an otitis media. Under such conditions the organic iron preparations, combined with malt and used in connection with or alternating with cod-liver oil, are of great value. It should also be remembered that following severe cases of scarlet fever some months are required for the heart to recover its normal tone, and in those cases where the rapid pulse continues the child should lead an outdoor life, but it should be carefully supervised as to the amount of exercise it takes.

Septic rhinitis, otitis media and other septic processes following scarlet fever may be successfully treated by autogenous vaccines. Kolmer and Weston have used vaccines very successfully in the treatment of septic rhinitis, and, since they found that the staphylococcus aureus was the cause of this condition in 89 per cent. of the cases which they examined, they recommended the use of the stock vaccine of this organism when it is not possible to obtain an autogenous vaccine. The initial dose of this vaccine is 50,000,000, gradually increased to 100,000,000 dead staphylococci.

## CHAPTER XL

### MEASLES, RUBELLA, AND ERYTHEMA INFECTIOSUM

### MEASLES

Measles is an acute infectious disease characterized by fever, catarrhal symptoms, an enanthem and an exanthem.

**Etiology.**—The specific cause of measles has not been discovered. Goldberg and Anderson produced the disease in monkeys by inoculating them with the blood of a measles patient. The infective microorganism multiplies rapidly in, and is readily disseminated from, the human organism. It does not, however, develop in other organisms or in outside culture media. Measles is more contagious, and is desseminated more rapidly in a susceptible community than any other acute infection, smallpox and influenza possibly excepted. Its spread, however, depends in a large degree upon rather close human intercourse; that is to say, by the well coming in contact with the sick in homes, schools and public gatherings. Notwithstanding the extreme contagiousness of this disease, it is not readily carried from the sick to the well by a third party, nor is the contagion in any other manner very readily carried long distances. It may, however, be disseminated through

the air of the room, and it is a recognized fact that there is greater difficulty in protecting other susceptible individuals in the same household by rigid quarantine than there is in scarlet fever or diphtheria. The contagion of this disease, unlike that of scarlet fever or diphtheria, has a short life outside of the human organism, so that the room recently occupied by a measles patient soon purifies itself. Measles is a world-wide disease. It is more prevalent during cold weather because of the closer indoor human intercourse during this season.

Nursing infants under six months of age are practically immune, but thereafter the susceptibility increases until, at the end of the first year of life, measles is not uncommon. The age of greatest susceptibility is between three and seven. During this period perhaps 90 per cent. of all children exposed contract the disease. This great susceptibility and the extraordinary contagiousness are the reasons why such a small percentage of the population escapes. Susceptibility diminishes very slightly with age. The chief reason why measles is largely a disease of childhood is because most adults have been rendered immune by an attack in early life. In large cities extensive epidemics recur every two or three years; this is due to the fact that in this time large numbers of children have grown up to the susceptible age since the last epidemic.

**Immunity.**—Natural immunity is very rare; perhaps not more than 10 per cent. escape. A permanent and lasting immunity is conferred by an attack of measles. While second and even third attacks have been observed in the same individual, it is rare indeed to find instances where this acquired immunity does not protect throughout life. This protection is more marked in measles than in any other of the acute infections.

**Period of Contagion.**—Measles is contagious from the beginning of the catarrhal stage until the end of desquamation. It is most contagious during the height of the fever and during the stage of eruption, but is generally spread by patients in the catarrhal stage, before the diagnosis has been made and the quarantine instituted. With the fall of the fever and the disappearance of the rash the contagion gradually diminishes, but probably lasts through desquamation. Ten days or two weeks later all contagion has disappeared, even though the sick room and belongings of the patient have not been disinfected.

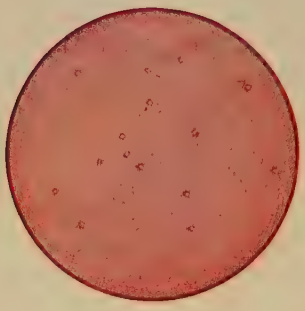
**Pathology.**—Measles is rarely a direct cause of death. It has, however, a comparatively large mortality, due to complicating conditions, such as enteritis, pneumonia, tuberculosis and, rarely, nephritis. Enterocolitis and pneumonia are especially dangerous in young children. The pathological changes belonging to measles proper are hyperemia of the skin and catarrhal inflammation of the mucous membranes of the respiratory passages and eyes. Other mucous membranes may also be affected.

**Incubation Period.**—This has been definitely established. The catarrhal symptoms occur in ten or eleven days, and the skin eruption in fourteen days from the date of exposure to the contagion. The period of incubation is marked by no characteristic symptoms; certain transitory rashes, such as

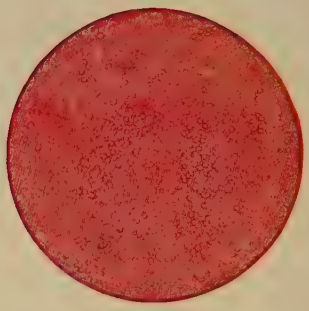
erythema and urticaria, may appear, but they are of little importance, and the child remains well until the catarrhal symptoms begin to develop on or about the eleventh day.

**Symptomatology.**—The symptoms may be conveniently divided into three stages: the enanthem stage, the exanthem stage and the stage of convalescence.

Enanthem Stage.—This is the stage of invasion and commonly lasts three days. Previous to the discovery of the characteristic enanthem of this disease by Koplik, a diagnosis during this stage was difficult and uncertain. The onset is usually marked with fever, drowsiness and catarrhal symptoms on the part of the eyes and respiratory passages. Irritation of the throat, coryza, sneezing and a beginning catarrhal irritation of the conjunctiva are important symptoms. Lacrimation and photophobia are commonly present, the patient shielding his eyes from the light. The cough which accompanies the bronchial catarrh is dry, harsh, and paroxysmal, and if the larynx be especially involved in the catarrhal process it is hoarse and croupy. These catarrhal symptoms increase in severity, the child becomes more languid, irritable and uncomfortable, and the fever, which is commonly remittent during this stage, increases from day to day. The appearance of the above symptom group, while not at all pathognomonic, should always suggest to the physician the possibility of measles. If to this there be added the history of exposure to contagion, a probable diagnosis can be made, but an early positive diagnosis can, as a rule, only be made by examining the mucous membrane of the mouth and there observing the characteristic enanthem. This is best described by Koplik himself: "On looking at the mucous membrane lining the cheeks, *in strong sunlight,* a very characteristic eruption of irregular stellate or round rose-colored spots is seen. In the center of each spot there is a bluish-white speck. This appearance of a bluish-white speck on a rose-colored background is pathognomonic of the onset of measles. The speck is sometimes so minute that strong sunlight is necessary to render it visible. The number of specks at the outset may be less than half a dozen. In a short time they become more numerous and the rose-colored spots become confluent so that there are diffusely red patches of buccal mucous membranes, studded with bluish-white specks. The specks rarely or never become confluent; their color does not resemble that of sprue, nor are they as coarse as sprue accumulations. They are seen on the inner surface of the lips and are sometimes well marked on the buccal mucous membrane adjacent to the gums of the upper molar teeth. If the finger is passed over the mucous membrane they are felt to be raised and firmly adherent. They can be rubbed off by force, or picked off with forceps. As the exanthema spreads, the enanthema of the buccal mucous membrane becomes diffuse. When the exanthem is at its height and during efflorescence the eruption on the mucous membrane begins to lose its characteristics. The bluish-white specks are washed away by the buccal secretions and leave the mucous membrane diffusely reddened and raw." Koplik's observations as to the



A



B

THE BUCCAL ERUPTION OF MEASLES (KOPLIK'S SPOTS). (HOLT).

A. This represents the earliest stage; the spots are few, rather large, widely separated, and usually show a distinct areola; the mucous membrane is normal in color.

B. The later appearance and that most frequently seen.

Near the center of the field the spots are closer together, although still remaining individually distinct; the mucous membrane is somewhat congested. At the margin of the field they are fainter and lack the areola, representing a still later period, such as is seen before they disappear altogether, although in some cases they are not more distinct than this at any stage.

diagnostic value and prevalence of this enanthem have been confirmed by the medical world. "Koplik spots" occur before the appearance of the skin eruption in 90 per cent. of the cases. They may usually be seen from twenty-four to thirty-six hours before the exanthem appears and in some cases earlier. In a few instances they are delayed and are coincident with the skin eruption. It is important to remember that they cannot be seen by artificial light, strong sunlight being necessary for their detection. Prior to the appearance of Koplik's enanthem, or coincident with it, there may be seen on the velum of the palate and on the mucous membrane of the hard palate a diffused redness, with deeper red or rose-colored spots, in the center of which are small white follicles. This enanthem was described by various writers before Koplik made his observations, but they are not so characteristic and have not the diagnostic value of "Koplik spots."

Exanthem Stage.—The fever, which, during the first three days of the disease, gradually increases in severity, reaches its height and continues high during the stage of eruption, not beginning to decline until the exanthem is fully developed. With the fading of the exanthem the temperature falls rapidly.

The exanthem or skin eruption, which is the characteristic symptom of this stage, commonly begins about the fourth day of the disease upon the face or behind the ears; it may first appear upon the back. It then spreads, gradually involving the neck, chest, back, arms, lower portion of the body, thighs, and lastly the hands and feet. Two or three days are usually required for the eruption to reach its height, but in some instances it may spread rapidly, covering the body within twenty-four hours, and in others it may be delayed, not reaching its maximum for four or five days. It appears first in the form of small red papules, about the size of a pin's head. These red points, which may be readily felt, are quickly surrounded by a small red zone, producing the typical macule of measles, which is round, oval or crescent shaped, and is still separated from neighboring spots by faint areas of normal skin. These macules grow darker in color and still further coalesce, forming darker patches, but in these larger zones of redness the small, dark-red, hard papules which formed the nucleus of the original eruption may still be seen and felt. However extensive the eruption of measles may become, it usually maintains its mottled, macular type, small areas of normal skin showing here and there throughout the eruption. The hyperemic character of this eruption is shown by the fact that it fades on pressure. This exanthem is one of the most characteristic of all eruptions, and when it occurs in a typical form can hardly be mistaken for any other rash. No description, however, can present to the mind a very clear picture of this or any other eruption. When the physician has observed the typical measles exanthem and noted its characteristics as above outlined he will ever after recognize it. Variations in the eruption are not uncommon. It may, in rare instances, be so mild and evanescent as almost to escape attention, and again it may rarely occur as an hemorrhagic, petechial rash, covering the entire body. In this latter form, known as "black

measles," hemorrhages from mucous membranes may occur and the disease, especially in infants, not uncommonly has a fatal issue. Between the mild and the hemorrhagic types we may have every grade of severity, but the great majority of cases conform to the ordinary type as previously described. With the spread of the eruption, all of the catarrhal symptoms noted in the enanthem stage are greatly exaggerated. The conjunctival irritation, photophobia, coryza, bronchitis, cough, rapid breathing, fever, and nervous symptoms are all increased. Young children may have convulsions, older ones are nervous, irritable, sleepless, and sometimes delirious. This aggravation in the symptom group continues until the eruption reaches its height, on or about the sixth or seventh day, when, rather suddenly, there is marked improvement in the whole symptom-complex. The temperature begins to fall and may reach normal in one or two days. The eruption fades rapidly, the nervous symptoms disappear, and the child becomes comfortable, passing into a restful sleep; the bronchitis and its accompanying cough gradually improve, and the exanthem stage has been transformed into the stage of convalescence.

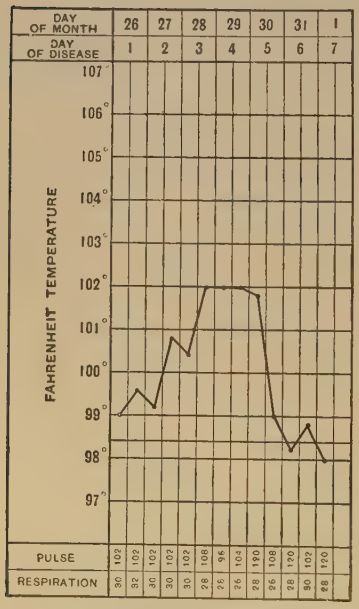



Fig. 55.—Measles Uncomplicated.

Stage of Convalescence.—The stage of convalescence lasts for a week or ten days. During this time the bronchitis, conjunctivitis and other catarrhal symptoms gradually disappear. The patient's appetite returns and he soon becomes impatient of the restraint which his quarantine entails. Desquamation begins with the fading of the eruption and continues for a week or ten days; it consists of small, fine, epithelial scales.

The Blood.—During the preëruptive stage there is a marked leukocytosis which falls to or below normal during the period of eruption. The polymorphonuclears are increased during preëruptive and eruptive stages; they fall below normal as the constitutional symptoms subside and return to normal during convalescence. The small lymphocytes are decreased during the height of the toxemia and increased as the symptoms begin to subside. The large mononuclears are increased late in the disease. The eosinophiles are decreased early in the attack and increased later.

The Urine.—The urine during the febrile stage is scant, highly colored and may contain traces of albumin. Acute Bright's disease, however, is a very rare complication. The diazo-reaction occurs in nearly every case of measles (80 to 90 per cent.). Acetone and diacetic acid may be found.

**Complications.**—*Bronchopneumonia* is the most common and the most

dangerous of all complications. It occurs most frequently in hospital wards, where staphylococci, streptococci and pneumococci abound, and is seen most commonly in children under four years of age; it is at this time of life that bronchopneumonia is so dangerous. Henoch says that every fatal case of measles shows some pneumonia. *Lobar pneumonia,* due to a complicating pneumococcus infection, is not uncommon in older children, but the prognosis in this condition is, on the whole, favorable. *Membranous laryngitis* may be a complication of measles. In some instances this pseudo-membrane may be produced by cocci, but for clinical reasons it is safe to assume that it is always diphtheritic. While membranous laryngitis is rather an uncommon complication of measles, a severe spasmodic, catarrhal laryngitis, producing pronounced croupy symptoms, is not

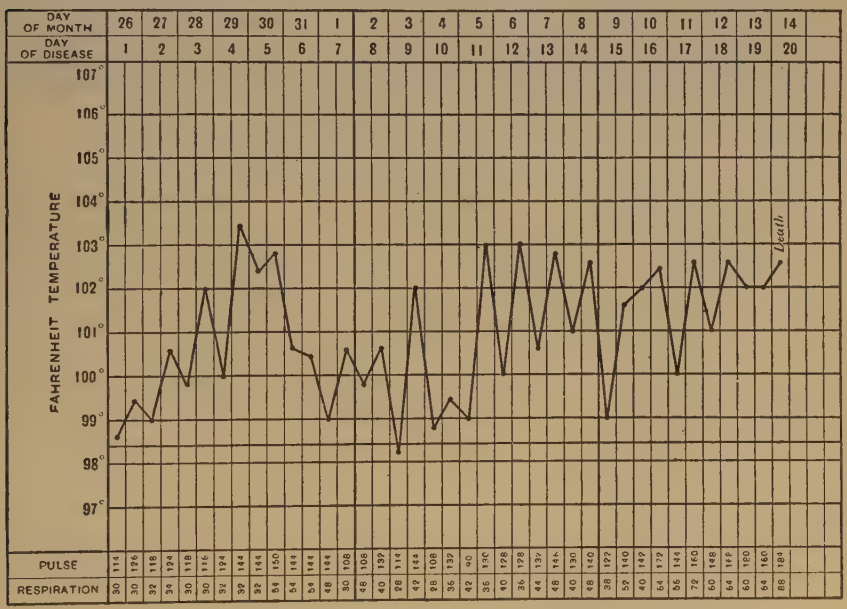



Fig. 56.—Measles Complicated by Bronchopneumonia.

at all uncommon. *Tuberculosis* is perhaps after all the most important complication of measles. In the chapter on Tuberculosis the remarkable prevalence of lymph-node tuberculosis is dwelt upon. The majority of children entering public hospitals have bronchial lymph-node tuberculosis, and this is one of the explanations for the frequency of bronchopneumonia among this class of patients. A large percentage of the bronchopneumonias occurring as a complication of measles is tuberculous, hence the importance of recognizing the fact that measles, which irritates the bronchial lymph nodes, is a disease especially liable to develop a latent into an active tuberculosis. General miliary and meningeal tuberculosis are not uncommon sequels of measles, and tuberculous pleurisy and empyema may occur.

Measles may irritate and inflame the mucous membrane of the gastrointestinal canal, in some instances causing a violent *enterocolitis.* This complication occurs especially in young children and may be serious, even fatal. *Aphthous stomatitis* and thrush may occur in young children, and their importance is increased by the fact that they must be differentiated from the enanthem of measles. *Conjunctivitis,* one of the catarrhal symptoms of measles, may become aggravated and produce corneal ulcerations and inflammations of the lacrimal glands. *Pemphigoid cutaneous eruptions* are rare and interesting complications, noted by many writers, but do not mean an unfavorable prognosis. Measles may also be associated with *whooping-cough, scarlet fever* and *diphtheria.* These mixed infections usually occur in institutions. The combination of whooping-cough and measles frequently results in a fatal bronchopneumonia; diphtheria

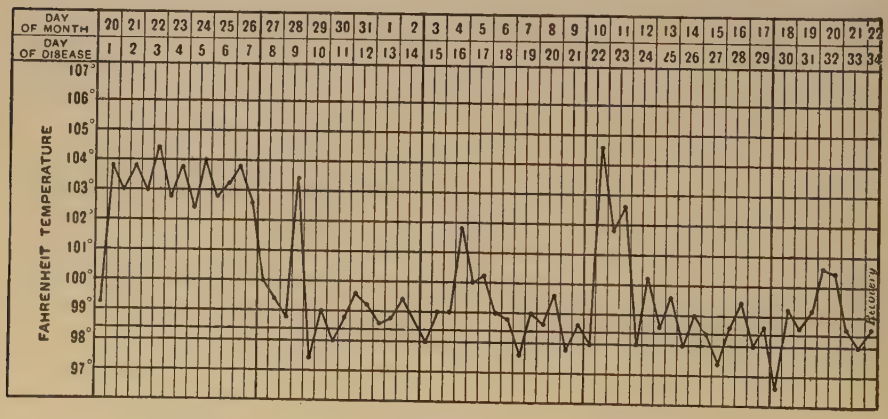



Fig. 57.—Measles Complicated by Bronchopneumonia.

and measles commonly mean membranous croup; scarlet fever and measles may cause a septicopyemia.

**Prognosis.**—The prognosis of uncomplicated measles in private practice is very good; nearly all such cases recover. In institutions it is very different; the death rate here, on account of the prevalence of dangerous complications, may be very high. Age is a most important prognostic factor. Holt places the mortality of this disease under two years of age at 20 per cent., while the total mortality is only about 5 per cent. The prognosis is also influenced by the character of the epidemic. If the disease is prevailing in an unusually severe or malignant form the prognosis is much graver than it is in ordinary epidemics. The previous condition of the child, especially the presence of lymph-node tuberculosis, greatly increases the danger. The various complications previously noted add a gravity to the disease which belongs to the complication and not to the measles.

**Prophylaxis.**—It is of the very greatest importance that young and tuberculous children should be protected from the contagion of measles.

To accomplish this a rigid quarantine should be instituted. If these two classes of children can be protected until they are older, they will be in a much better physical condition to withstand this disease. The laity, however, cannot, as a rule, be brought in sympathy with rigid quarantine measures in measles. They argue with some degree of truth that the child must have it some day and why not now, when it can be properly cared for. Whether or not the physician takes this view of the case with the normal healthy child over four years of age, there is no question as to his responsibility in the matter of protecting the very young and the tuberculous, and in all instances it is his duty to insist upon a quarantine that will at least protect people outside the family. The young and tuberculous are better protected by sending them away from home when it is not possible to send the patient away. Following convalescence, the sick room and all of its belongings should be disinfected with formaldehyde and thoroughly ventilated and cleaned. This effectively destroys the contagion.

**Treatment.**—Measles is a self-limited disease for which we have no specific medication. Treatment should therefore be directed to the relief of uncomfortable symptoms and the avoidance of complications. The patient should be put to bed and kept there until convalescence is established. The bedroom should be well ventilated and kept at a temperature of about 70° F. Sudden chilling of the surface of the body by great variations in the temperature of the room, or by draughts of cold air, or baths with cold water, are to be avoided. The patient should be sponged off with warm water once a day, and afterwards rubbed with lanolin or oil. If he complains of itching, carbolic acid may be added to the lanolin. The room may be slightly darkened, but light should not be excluded. The patient's eyes, especially if he suffers from photophobia, may be shaded.

The DIETETIC TREATMENT is important, especially in young children. All children under three years of age should be fed during the onset of the disease as though they had an enterocolitis. Barley water, broth, diluted skimmed milk, and peptonized milk are among the foods especially suitable to ward off gastrointestinal complications. In older children a light, simple diet, having milk for its basis, should be employed during the acute stage. A moderate amount of cool water may be taken, and where the throat symptoms are especially irritable small bits of ice in the mouth are permissible. The prejudice of the laity against cold drinks and cold baths may, to a certain extent, be respected without inflicting great punishment on the patient; but the hot bedroom, hot drinks and heavy bed clothing, for the purpose of "bringing out" the eruption, are superstitions which may be resisted, greatly to the comfort of the patient.

SYMPTOMATIC TREATMENT.—The *cough* and *bronchial irritation* usually require treatment. In younger children the bromid of potash or soda, combined with small doses of belladonna, put up in essence or elixir of pepsin, usually allays the cough; if this does not suffice, small doses of chloral may be added to this mixture. In older children it may be advisable to control the irritable cough with small doses of codein or pare-

goric, but it should be remembered that these opiates are in no sense curative and their effect should therefore be carefully studied; if they benefit the patient more than they do him harm, they may be continued. These drugs produce constipation and interfere to a certain extent with the appetite and digestion. Syrups and expectorants should never be used. I do not believe any good whatever can result from the syrup of ipecac, syrup of squills and ammonia preparations commonly used, and I know that they may do much harm by disturbing the appetite and digestion. They should at least never be given to young children. The *fever* and *nervous symptoms* may be controlled by sponging with lukewarm water, and by the giving of antipyrin, aspirin and phenacetin in doses suited to the age of the child. I do not believe any harm whatever results from the use of antipyrin associated with tincture of strophanthus and combined in a suitable elixir, and I do believe that this prescription, when given in proper doses to suit the age of the child, will very materially relieve the distressing symptoms and thereby add to the comfort of the patient, during both the enanthem and exanthem stage of this disease. The following prescription may be used:

℞ Antipyrin ..... ℥ ss
Tinct. strophanthus ..... ℥ ss
Tinct. belladonnæ ..... ℥ ss
Elixir lactated pepsin ad..... ℥ iii
Teaspoonful every four hours for a child four years of age.

If *lung complications* develop, guaiacol inunctions and carbonate of guaiacol internally should become a part of the routine treatment. With the onset of *septic symptoms* and especially *pneumonia,* the guaiacol ointment is changed for unguentum Credé and a number of doses of antistreptococcic serum given, as outlined in the treatment for scarlet fever. In older children inhalations of creosote and oil of eucalyptus may be used as a bronchial antiseptic, and to relieve the symptoms resulting from respiratory irritation. No medicines are necessary to bring out the *eruption,* but, in cases where it has failed to come out properly and a *bronchopneumonia* is threatening or has already begun, warm mustard baths may be of value in increasing the peripheral circulation and relieving the pulmonary congestion. These cases may also be benefited by applications of warm camphorated oil applied under an oil silk jacket, as outlined in the Treatment of Bronchopneumonia. Cathartics are to be used cautiously in the treatment of measles. Enemata commonly suffice to keep the bowels open; when necessary, however, a mild cascara laxative may be used. If *enteritis* threatens castor oil should be given and the patient placed on the diet outlined under Enterocolitis. Pneumonia, tuberculosis, membranous laryngitis and other complications are to be treated as elsewhere noted. In *membranous laryngitis* it is always safest to administer diphtheria antitoxin, even though there may be a possibility that the pseudomembrane is produced by cocci. The *eyes* should be properly protected

during convalescence, and their use for reading and other close work should be prohibited until all traces of conjunctivitis have disappeared.

## RUBELLA

*(Rötheln, German Measles)*

Rubella is an acute infectious disease in no way related to ordinary measles. It is characterized by slight fever, enlargement of the post-cervical lymph nodes and by a rash resembling in some particulars both that of measles and scarlet fever.

**Etiology.**—Breast-fed babies under six months of age are practically immune. During the latter half of the first year of life the susceptibility to this disease gradually increases. From that time on nearly every individual is susceptible. Age does not confer immunity. Adults and children alike nearly always contract the disease when they come in close contact with the infection. Epidemics are much more common during the winter months.

The specific microorganism of rubella is unknown, and like the contagious principle of measles it is short-lived after it leaves its host. The contagion does not, for any length of time, cling to and contaminate the clothes, bedding and other belongings of the patient. It is spread by the well coming in contact with the sick; rather close contact is necessary. There is little or no danger of a third party carrying the contagion from the sick to the well. The infection, however, may be carried short distances through the air and by fomites. There is great variability in the contagiousness of this disease in different epidemics. The period of contagion begins a day or two before the eruption appears and lasts until it disappears.

**Immunity.**—One attack confers immunity; second attacks are very infrequent. An attack of this disease does not afford protection from measles or scarlet fever, nor do these diseases protect the patient from rubella. Rubella, measles and scarlet fever may follow each other in rapid succession as house epidemics in the same family.

**Incubation.**—The period of incubation varies from ten to twenty days. Griffith reports a case where it was one day. It would appear, therefore, that the incubation period is either very variable, or that more accurate observations are necessary to establish its limits.

**Symptomatology.**—The ONSET is commonly mild. The rash may be the first symptom noted. It may be accompanied or preceded by a slight elevation of temperature, a feeling of general malaise, headache and backache. There may be mild catarrhal symptoms, such as slight pharyngitis, coryza and infection of the conjunctiva. A slight cough and gastric disturbance may be present. The fever does not run high. In some instances it may scarcely be above normal. In severe cases, especially in young children, it may reach 103°F. Its maximum is commonly attained

within the first thirty-six or forty-eight hours, and it then subsides. There is nothing at all characteristic in the fever, pulse, or respirations.

The ENANTHEM of this disease is always present and is a valuable aid in differentiating it from other acute infections. Forchheimer says of this enanthem: "It consists of a macular, distinctly rose-red eruption, upon the velum of the palate and uvula extending to, but not on, the hard palate. The spots are arranged irregularly, not crescentically, of the size of large pin-heads, very little elevated above the mucous membrane, and do not seem to produce any reaction upon it." The enanthem appears just before or simultaneously with the exanthem or skin eruption, and lasts one or two days. The whole mucous membrane of the throat, especially the pharynx, is red and congested.

The EXANTHEM, or skin rash, is of special value in diagnosis when it is associated with Forchheimer's enanthem. The skin rash in and of itself is very puzzling from a diagnostic standpoint, because of its variations. It commonly appears as small, pale rose-red spots somewhat smaller than the measles macule. This rash, however, resembles to a certain extent the measles eruption, but is lighter in color. These macules may coalesce as in measles, forming patches of rose-red colored skin. In other instances the rash may occur as fine, red points producing more or less uniform redness of the skin. This type of the eruption, therefore, more closely resembles the exanthem of scarlet fever. It has not, however, the scarlet hue and can usually be differentiated from scarlet fever by the rose tint and less punctate form of the rash. Both forms disappear on pressure. In some cases the two eruptions occur in the same patient; on one portion of the body the macular measles-like eruption predominates, and on another the uniform rose tint, somewhat resembling scarlet fever, may be seen. The exanthem begins on the face and spreads downward, involving the neck and body and lastly the arms and legs. A point of diagnostic importance is that the eruption, unlike that of scarlet fever, appears on the lips close around the mouth. Another point of great diagnostic importance is that the rash, unlike those of measles and scarlet fever, appears on the first day of the disease, reaches its maximum by the second day, and then begins to fade. It may entirely disappear in one or two days, or traces of it may linger for a week or ten days. The severity of the disease is not measured by the length of time the rash continues. It may disappear and again return; this, however, is very uncommon.

The IRRITATION OF LYMPHATIC TISSUES is very characteristic of this disease. The spleen in almost every instance is slightly enlarged, and can be palpated. Enlargement of the posterior cervical lymph nodes is a constant and distinctive feature, since they are not so uniformly enlarged in any of the other acute infections. The nodes in the neck most commonly enlarged are the post-cervical, the suboccipital and the post-auricular. The anterior cervical nodes, however, may also be enlarged and the superficial lymph nodes throughout the body can commonly be felt.

The BLOOD shows a polynuclear leukocytosis during incubation and leukopenia during the stage of eruption.

THE URINE.—The diazo-reaction, which is so constantly present in measles, is absent in this disease.

**Complications.**—The lighting up of a latent tuberculosis is the most common complication of rubella. Other complications are rare. In the more severe cases gastrointestinal and respiratory diseases may occur.

**Prognosis.**—This is good. Fatalities have been reported in epidemics of extreme severity.

**Treatment.**—The patient should be isolated until a positive diagnosis has been made. It is a matter of serious import to mistake a mild scarlatina for rubella. The diagnosis once established, the disease is so simple as not to require a rigid quarantine. Patients, however, should be confined to their homes, so as to prevent spreading the disease.

The average patient requires no medical treatment. In the more severe cases a mild laxative should be given, and the patient confined to bed for one or two days. Small doses of antipyrin and phenacetin may be given to relieve the fever and nervous symptoms.

## ERYTHEMA INFECTIOSUM

Erythema infectiosum is an acute infectious disease first described by Escherich. It is characterized by a rose-red rash with slight or no constitutional symptoms.

**Etiology.**—This disease is believed to be rare in America, although in recent years small epidemics have been observed in our larger cities. Infants under one year of age are immune. Older children are commonly affected, and adults are susceptible. It usually occurs in epidemics. The specific organism is unknown. It is spread, however, by contagion, rather close contact being necessary, and it is in no way related to rubella, scarlet fever or measles.

**Symptomatology.**—After an incubation period of ten or twelve days an erythematous eruption may appear on the face. This rather brilliant red rash spreads over the cheeks onto the body. Parts of the skin, however, are commonly normal in color, giving the eruption a blotchy appearance. It is a typical erythema, having much the appearance of erysipelas, except that the skin is not inflamed as in that disease. The exanthem lasts for about one week, gradually fading, and is not followed by desquamation. The patient may suffer from slight headache and sore throat. The temperature rarely rises above 100°F., so that this condition is practically an afebrile disease. The lymphatic tissues are not involved as they are in rubella.

**Prognosis.**—This is always good, and no treatment is required.

The following table from Ruhräh will assist in the differentiation of the acute exanthemata:

## Differential Diagnosis of Rubella, Scarlet Fever, Measles and Erythema Infectiosum

| | Rubella | Measles | Scarlet Fever | Erythema Infectiosum |
|---|---|---|---|---|
| Contagion | Apparently varies in epidemics. Direct contact. Possibly from fomites, not through the air. | Highly contagious. By direct contact. By fomites. Through the air. | Marked. By direct contact. By fomites. | Feeble. Usually by direct contact. |
| Incubation. | Variable average 1 to 3 weeks. | Average 9 to 14 days. | Average 1 to 6 days. | Average 6 to 14 days. |
| Prodromes. | Slight and of short duration. Occasionally a day or two of malaise. | 3 to 4 days. Drowsiness and catarrhal symptoms. | Short or wanting—onset usually sudden. | Very slight and of short duration. |
| Koplik spots. | None. | Present in 90 or 95 per cent. of cases. | None. | None |
| Vomiting. | Rare. | Occasional. | Common. | Uncommon. |
| Fever. | Slight—average 1 to 2 days, sometimes for 4 days, seldom more than 101° to 102°. | Marked high curve lasting about a week, average from 102° to 104°. | High fever lasting about a week, average 104° to 105°. | Little or none. |
| Catarrhal symptoms | Slight. | Marked. | Absent. | None. |
| Tongue. | Slight coat, nothing characteristic. | Tongue coated, that of any fever. | Strawberry, later mulberry tongue. | Sometimes slightly coated. |
| Throat. | Small, punctiform, red spots over uvula and palate. Pharynx slightly reddened. | Moderate pharyngitis and redness of mucous membranes | Usually a severe angina. | Sometimes very slight sore throat at onset. |
| Diarrhea. | | Frequent. | | |
| Lymph nodes. | General enlargement especially of postcervical nodes. | Postcervical, postauricular, and submaxillary nodes enlarged. | Depends on extent of throat involvement, glands at angle of the jaw involved. | Not enlarged. |
| Pulse. | Varies with fever. | Varies with fever. | Very rapid | Normal. |
| Albuminuria. | Rare and slight. | Rare. | Common. | None. |
| Eruption. | Begins on face, spreads to neck and breast, then to arms, legs and feet. Is fading from older parts while spreading to new. Two forms—common form, morbilliform, small, slightly elevated papules, discrete, sometimes confluent; more rarely scarlatiniform, lasts 2 to 4 days or less, color rose-red but this varies. | Begins on face, spreads gradually over entire body, covering it by the second or third day. Consists of small papules arranged in crescentic groups; these are confluent in places. Lasts 4 to 5 days. Is deep red, often purplish. | Begins on neck and chest, spreads slowly over entire body—maximum about the fourth day. Does not affect lips. Consists of small, punctate spots or a diffuse blush; disappears on pressure; lasts about a week. Intense red color. | First on face as symmetrical, rosered blush, for the most part sharply defined and resembling erysipelas. It is hot to the touch but not sensitive and it does not itch. The second day it spreads to the body and extremities, small discrete crescentic patches over the body and sparingly on the inner and flexor surfaces of limbs. Marked map-like eruption on outer and extensor surfaces. Begins to fade on face in 4 or 5 days. Lasts altogether 6 to 10 days. |
| Desquamation. | Slight and branny. | Branny. | Marked in flakes and large pieces. | None. |
| Convalescence. | Rapid, no complications. | Slow, frequent complications, as pneumonia. Later other infectious diseases, as tuberculosis. | Slow, complications frequent, as nephritis, otitis media, etc. | Rapid, no complications. |

# CHAPTER XLI

## VARIOLA, VACCINIA AND VARICELLA

## VARIOLA

Variola, or smallpox, is an acute infectious and highly contagious disease characterized by more or less severe constitutional symptoms and by a specific eruption passing through the stages of papule, vesicle, pustule, scab and scar. The disease has no peculiar manifestations in childhood, running much the same course at all ages. Before the days of vaccination the disease was almost confined to childhood, 90 per cent. of the cases occurring in children under ten years of age. The immunity of adults during that period was due to the fact that a vast percentage of the adult population was immune from having had the disease. At the present time, however, by reason of the fact that nearly all children are vaccinated, the disease is rarely seen in childhood. It is now, therefore, in civilized communities, a comparatively rare disease confined almost exclusively to adults, among whom the immunity which resulted from early vaccinations has wholly or partially expired. The disease is so infrequent at the present time that it is rarely seen in private practice. Many physicians with a long and active medical career have never come in contact with it.

**Etiology.**—The specific microorganism of smallpox is now believed to be the "Cytoryctes Variolæ," a parasitic protozoa, first clearly described by Guanieri in 1892, and subsequently shown to be etiologically related to this disease by Councilman and his associates.

**Contagiousness.**—It is the most highly contagious of all infectious diseases, being spread directly by contact of the sick with the well, and indirectly by the contagion being carried by a third party, and by clothing, bedding, and other belongings of the sick room. The contagion is given off from the lungs, and by the discharges from the vesicles and pustules of the skin eruption. The dry crusts may carry and transmit the disease long distances. It is believed that it may be transmitted through the air for distances sufficient to make house to house contamination possible. The contagion lasts from the beginning to the end of the disease, but is very slight until the skin eruption appears. Race and sex offer no barriers to its dissemination. The cold winter months furnish conditions favorable to its spread. Age does not confer immunity, but children are slightly more susceptible than adults.

**Incubation.**—Smallpox has a very definite period of incubation; this is usually ten to twelve days, and it is believed that its limits are from five to twenty days.

**Symptomatology.**—The Stage of Invasion.—The onset is marked by very pronounced symptoms. Headache, backache, convulsions, gastric dis-

turbance and profound depression are present in typical cases. In older children and adults a chill takes the place of the convulsion. There may be great variation in the severity of this initial symptom group. Occasionally severe cases have a mild onset, but, as a rule, the initial toxemia is pronounced; this is especially so in unvaccinated children in whom the nervous system is profoundly affected. Not infrequently convulsions, stupor, coma, delirium and profound depression occur. The stage of invasion lasts for about three days, during which time severe backache in the lumbar region is a rather characteristic symptom, and the headache may continue to be so severe that therapeutic measures are required for its relief. The initial vomiting may not be repeated, but loss of appetite and gastric discomfort are present. The fever rises rapidly with the initial chill; it may reach 104° or 105°F. within the first twenty-four or thirty-six hours. It usually reaches its highest point on the second or third day, and then, in the less severe cases, falls rapidly to normal, and even in severe cases there is a sharp fall in temperature. After this fall there may be only a slight elevation of temperature during the next few days, when there is a secondary rise coincident with the formation of pustules. This septic or secondary fever continues for a number of days, during the pustular stage, and then falls to normal as convalescence approaches. The height and duration of the secondary rise in temperature will depend upon the severity of the infection and the character of the eruption. During the stage of invasion cutaneous eruptions may occur in a small percentage of cases. Both erythematous and petechial rashes are seen; the latter, which occur about the second day, when associated with the symptoms above noted, are of diagnostic importance.

The ENANTHEM is one of the most valuable of diagnostic symptoms, as it commonly occurs from twelve to twenty-four hours before the skin eruption. It consists of small red papules which can be distinctly seen and felt on the hard and soft palate, and may occur on any portion of the mucous membrane of the mouth. These papules become vesicles, and, if not ruptured, pustules; the rupture, however, usually occurs during the vesicular stage. As an examining physician to a large general hospital many years ago, I had the opportunity of testing the value of the enanthem in making an early diagnosis of these cases. During the epidemic of smallpox then prevailing every patient applying for admission to the general hospital was carefully examined for the smallpox enanthem and in many cases the diagnosis was made upon this sign and the patient sent to the smallpox hospital, to break out the next day with a typical skin eruption. The importance of making the diagnosis at this early period is greater because the time of great contagiousness begins with the exanthem stage. Prior to this the disease is but feebly contagious.

STAGE OF ERUPTION.—About the third day the characteristic skin-eruption commences to make its appearance, and the pain in the back, headache, gastric disturbance and fever quickly subside. The eruption first shows itself as small red papules, which can be felt as well as seen;

as they increase in size they give to the skin a peculiar shotty feel. On the third day the papule becomes slowly transformed into a vesicle filled with slightly grayish fluid; as the vesicle increases in size it becomes distinctly umbilicated and is surrounded by a small hyperemic zone. About the eighth or the ninth day the vesicle is converted into a pustule, the contained fluid having a yellow color. During this time as the pustule matures the surrounding skin becomes more indurated and inflamed, so that if the pustules are located near together, the whole surface becomes inflamed and indurated. The pustule maintains its umbilicated form for a number of days and then slowly begins to dry, forming a brown scab. This change commences about the twelfth day, and with it the surrounding skin becomes less inflamed; as the inflammation subsides the crusts become dryer and begin to drop off during the third week, leaving a red scar. General desquamation then begins and is usually completed about the end of the sixth week; in milder cases earlier, in more severe cases later. The smallpox eruption first makes its appearance on the face, then spreads to the trunk and later to the extremities. It is especially profuse around the neck and back, and not so marked below the knees and on the abdomen. As previously noted, there is a secondary rise of fever during the pustular stage, and as the eruption reaches its height, about the ninth or tenth day, the skin may become so swollen as to produce great pain. In severe forms of the disease, where the skin is very intensely inflamed, the pain is very acute; the location of individual pocks, as for example under the nail, in the auditory canal, and in the larynx, and in the eye, may greatly increase the suffering, and in the latter location may result in loss of sight.

The Urine.—Welsh and Schamberg found albuminuria in 50 per cent. of cases which recovered, and in 84½ per cent. of the fatal cases. Acute Bright's disease, however, is a rather rare complication.

Blood.—There is, as a rule, a marked leukocytosis, especially during the vesicular stage. The lymphocytes are increased and the polymorphonuclears decreased. The red blood cells are greatly diminished in the later stages of this disease.

**Clinical Forms.**—Variola may present itself in many forms. The mild cases have been termed varioloid. These commonly occur in vaccinated individuals in whom the immunity derived from the vaccination has partially run out. In these cases the constitutional symptoms are mild and the eruption very slight; only a few pocks may occur. From this mild type we have every grade of severity to the confluent form, in which the pustules are so close together that they become confluent, and the associated dermatitis is therefore very much aggravated. In these cases the constitutional symptoms are not only severe, but the pustular stage of the disease presents a very revolting picture; the patient's face and eyes being so swollen that he cannot be recognized by his best friend. Hemorrhagic smallpox is a very grave form, in which the pocks become hemorrhagic and hemorrhages occur from mucous membranes. The constitutional symptoms are profound and death

usually results. Purpuric smallpox is a fatal form in which petechial hemorrhages appear as early as the third day, taking the place of the ordinary eruption. Hemorrhages occur from mucous membranes and from the kidney and the patient dies, as a rule, before the characteristic eruption is developed.

**Diagnosis.**—Before the skin eruption appears, the diagnosis of smallpox may be made by the constitutional symptoms, the petechial rash, and the typical enanthem on the palate. After the skin eruption appears the only disease with which smallpox is likely to be confused is chickenpox, and the differential diagnosis of these two conditions is given in the chapter on that disease.

**Prophylaxis.**—Vaccination is the all-important measure in the prophylaxis of smallpox. The disease may be prevented in this way. This subject is discussed under Vaccination. The only other measure of any importance is the absolute and complete isolation of the patient, and this can only be done satisfactorily by removing the patient to a smallpox hospital. If he is treated at home the other members of the family should leave the house, and he should be given into the hands of the doctor and trained nurses. Under these conditions the most rigid quarantine possible should be established, even more rigid than that described under Scarlet Fever.

**Treatment.**—During the stage of invasion the patient is to be made as comfortable as possible by symptomatic treatment. For the fever, frequent spongings of the body with cool water should be resorted to if it adds to the comfort of the patient. An ice-bag to the head will modify the headache, the nervous symptoms, and help to reduce the temperature. Bromid of potash, chloral and, if necessary, sulphate of codein or morphin, in doses suited to the age of the child, may be used to relieve the intense suffering of this stage. Chloral and bromid of potash are especially valuable in young children for the control of the convulsions and other nervous symptoms, and if the stomach be so irritable that these drugs cannot be given by the mouth, then the chloral alone should be given by the rectum. The diet during the acute stage should consist of milk, cereals, bread and fruit juices, the object being to furnish nutrition without overtaxing the digestive organs or the kidneys.

With the secondary rise in temperature which occurs during the pustular stage of the eruption, active symptomatic treatment is again demanded to relieve the distress produced by the swelling and inflammation of the skin. The itching during this stage may be very great and the patient must be prevented from scratching. The tearing of the pocks on the face should be especially guarded against to prevent scarring. Cold applications offer the greatest relief; these may be made by wringing cloths out of ice-cold water and applying them as a mask to the face or to other portions of the body where the suffering is intense. Carbolic acid may be added to the water, as this helps somewhat to relieve the itching. In severe cases the eye demands careful attention, and as the conjunctivitis

becomes marked it is necessary to separate the lids and wash out the accumulated discharges with a weak boric acid solution. Cloths wrung out of ice-water should be applied to the lids. Pharyngitis should be treated by weak alkaline antiseptics. In rare cases the eruption extends to the larynx, threatening suffocation; this condition may demand tracheotomy. As smallpox in the unvaccinated is a very dangerous disease and one in which rather sudden collapse is not uncommon, active stimulation may be demanded at any time. For this purpose alcohol in the form of whiskey or brandy should be freely administered in combination with tincture of strophanthus or tincture of digitalis. The red-light treatment of smallpox is believed to exercise a favorable influence on the skin lesions. This can be used only in a specially prepared room where all the light is filtered through red glass.

## VACCINIA

Cowpox is believed to be modified smallpox, as it occurs in the cow; it manifests itself in a vesiculo-pustular eruption on the udder and teats. Vaccination with the virus of the cowpox vesicle communicates this disease to man, producing vaccinia and protecting him from the contagion of smallpox for a variable length of time.

**History.**—Before the time of Jenner it was known to dairy workers that an attack of cowpox conferred immunity from smallpox. In fact Jenner's attention was called to this subject by coming in contact with dairy people. After a careful investigation he made his first vaccination on May 14, 1796. This was done with virus taken from a milkmaid suffering from cowpox; the subject was a boy named James Phipps; subsequently with the same virus he vaccinated his own son, and a number of other children. Later he inoculated some of these children with the virus of smallpox and otherwise exposed them to the contagion of this disease, but none of them contracted it. Jenner continued his investigations over a period of two years, and in 1798 published his observations in which he stated that patients who had had cowpox were ever after protected from smallpox, and that smallpox could therefore be prevented by inoculating the patient with cowpox. Thus originated perhaps the greatest of all medical discoveries, *vaccination;* a discovery which has saved more lives than any other, and which has almost obliterated smallpox, the most terrible pest of the seventeenth and eighteenth centuries.

It is difficult for us at the present time to realize that the discovery of vaccination by Edward Jenner was and still remains the greatest of all triumphs of preventive medicine. Crandall, who has most carefully studied the vast literature of this subject, says: "A hundred years ago smallpox was justly regarded as the Attila of diseases, the very scourge of God, overrunning countries and destroying populations. When Jenner performed his first vaccination it was causing one-tenth of all the deaths of the human race. Bernouilli, the mathematician, estimated that more

than 60,000,000 of the inhabitants of Europe died of smallpox during the eighteenth century. Others place the number even higher. Specific proof of its fatality is shown by Cowan's vital statistics of Glasgow. In that city between 1783 and 1792, 36 per cent. of all deaths under ten years of age were due to smallpox. One-third of all the deaths in Europe under ten years were due to the same cause. When smallpox was introduced into Mexico by the Spaniards in 1520, 3,500,000 died within a few years. In 1737 in Iceland, 18,000 in a population of 50,000 died in a single year. It is believed that 6,000,000 North American Indians fell victims of its ravages." In contrast with this let me quote the opening sentence of a recent encyclopedic article on smallpox by Ch. Bäumler, of Freiberg: "There has been no opportunity of observing variola in any form in this clinic for nine years; still it is necessary for every well-informed physician to have a knowledge of this important disease. As a result of strict vaccination and revaccination in Germany, the disease has been prevented from appearing, so that many physicians have never had an opportunity of seeing smallpox." The results which have been attained by Germany could be obtained in other countries if compulsory vaccination were adopted. In the United States smallpox is kept alive by the fact that there still exists an ignorant negro and uneducated foreign population and a few fanatics who "do not believe in vaccination."

**The Vaccine Virus.**—In the early experiments vaccinations were made with the lymph taken directly from the cowpox vesicle. Later humanized virus came into very general use, the subject being vaccinated either with the fresh lymph from a human vaccine vesicle or with the dried lymph in the form of scabs or crusts which came from the matured vaccine vesicle. As time went on public opinion was much opposed to the use of humanized virus, because of the exaggerated views then prevalent of the danger of transmitting syphilis, tuberculosis and possibly other diseases. At the present time humanized virus is rarely used, bovine virus having taken its place. This vaccine virus is now prepared from the serum of the cowpox vesicle, and is put on the market in the form of a glycerinated bovine virus in hermetically sealed tubes, or on ivory points which are themselves protected from contamination by being put up in glass tubes or other coverings; the object being to present to the public a pure, sterile bovine virus which can be used without fear of infecting the patient with pathogenic organisms.

**Technique of Vaccination.**—Vaccination is to be performed under strictly aseptic conditions. The skin is to be carefully cleansed with soap and water and then with alcohol. The operation may be made with an ordinary sewing needle, or with the sterile ivory vaccine points above described. If the needle is used it should be sterilized by heating, and with its point the skin is to be carefully scratched four or five times, both in a longitudinal and transverse diameter, producing a raw surface about one-sixth of an inch square. The scarification should be deep enough to remove the superficial epithelium, leaving a red, raw, but not bleeding, surface.

Into this raw surface the vaccine virus is rubbed with the ivory vaccine point, or some other sterile instrument. The wound should then be allowed to dry before the clothing comes in contact with it; this commonly requires fifteen or twenty minutes. Following this operation the vaccine wound is to be carefully protected from traumatic injury and infection. The point on the skin usually selected for vaccination is the upper and outer surface of the left arm at or near the insertion of the deltoid. Another point of selection is the outer surface of the left leg, six or eight inches above or below the knee. Of these two locations the arm is by far the best. The leg, however, may be selected in young infants under one year of age, since at this time of life it is almost as easy to properly care for the vaccine wound on the leg as it is upon the arm. In children old enough to be upon their feet there is more or less danger that the vaccination wound on the leg may be injured and contaminated; for this reason first vaccinations in older children should always be made on the arm. If not, the parents should be made to assume the responsibility of keeping the child off its feet during the period of marked inflammation in and around the vaccine vesicle. For esthetic reasons the physician is very frequently requested to vaccinate girls upon the leg, as the scar upon the arm is unsightly. Vaccination should, if possible, be performed during the first year of life, but it is advisable to wait until after the child is three months of age, or until its nutritional problems are solved; it is then ready for vaccination, and the earlier the operation is made the milder will be the course of the vaccinia and the less trouble will there be in the care of the wound. If, however, the infant be tuberculous or come from a tuberculous family, or if it be malnourished from lack of proper food or other causes, vaccination may be postponed until these nutritional faults are corrected. In the meantime, however, should smallpox appear in the community, the child should be vaccinated without further delay.

**Incubation Period.**—The incubation period of vaccinia is from three to five days, that of smallpox eight to twenty days; the average incubation period of vaccinia is four days, that of smallpox twelve days. These are most important facts, since they explain why a patient who has been exposed to smallpox may even then be protected by prompt vaccination. If the vaccination be made within the first day or two after exposure to the smallpox contagion, the patient may escape smallpox, as the vaccinia reaches its height before smallpox has had time to develop. If the vaccination be made four or five days after exposure to the smallpox contagion, the vaccinia may still precede the smallpox in its development and cause it to run a very mild course.

**Clinical Manifestations.**—Directly following vaccination the wound heals and on the fourth or fifth day thereafter a faint red spot makes its appearance, which soon manifests itself as a papule with a red base. On the sixth or seventh day this papule becomes a vesicle of grayish color with a slight zone of redness and contains a clear sterile lymph. On the eighth

day the vesicle becomes contaminated with bacteria and the lymph becomes clouded with pus corpuscles, so that by the tenth day a pustule has taken the place of the vesicle. In the meantime as the pock develops from the vesicle into the pustule it becomes umbilicated, a slight central depression showing on the eighth or ninth day. During this time the zone of redness which surrounded the vesicle is much increased in size and encircles the pustule from half an inch to an inch or more in every direction; this zone is more or less thickened and indurated and, near the pustule, slightly tender to the touch. From the ninth to the eleventh day the disease is at its height, and during this period adjacent lymph nodes are swollen and tender. When the arm is the site of the vaccination the axillary lymph nodes are especially involved, and may appear as hard, tender tumors the size of a hickory nut. After the eleventh day the inflammatory process subsides, the umbilicated pustule begins to dry up and form a scab. The axillary lymph nodes diminish in size, the surrounding zone of redness and induration gradually diminishes, and all the acute symptoms disappear. The scab or crust does not usually fall off until the end of the third week, and may remain a week longer. During this period great care should be exercised to have it remain as long as possible, to be finally cast off by necrotic processes which have undermined it. With the falling off of the scab a depressed, red surface is left, which later marks the site of the permanent scar, which usually has a pitted appearance.

With the onset of other acute symptoms fever usually appears on the eighth or ninth day. In infants under one year of age the fever is very slight, and even in older children it does not commonly rise above 102° or 103°F.; it may, however, reach 104°F. Soon thereafter the temperature begins to fall and may reach normal within two or three days. The febrile reaction in vaccinia varies greatly. It may be very slight and evanescent, and it may be well marked and last for four or five days without indicating septic contamination of the wound or other complications. The typical lesion or pock of vaccinia in first vaccinations runs the same course as the lesion of smallpox. It is first a papule, then a vesicle, pustule, scab and scar. These stages, however, are milder and shorter in vaccinia than they are in smallpox, but they are characteristic of vaccinia, and a vaccine sore that does not present this sequence is abnormal, and may not afford protection against smallpox. The severity of the course of vaccinia does not increase its power of protection. The mild course which this disease runs in infancy confers immunity for as long a period as the more severe vaccinia of the older child. On the other hand, it should also be noted that vaccinia marked by severe local and constitutional symptoms is not to be considered abnormal, provided the disease runs the typical course above outlined. It simply means that some individuals are more susceptible to vaccinia, as they are to other contagious diseases.

Secondary Rashes.—In some instances a dark red swelling occurs at

the point of vaccination instead of the typical sore. This has been described as the "raspberry excrescence." It is firm, considerably elevated above the surface of the skin, but is not inflamed or tender, and has no discharge. This condition is brought about by some abnormality in the virus used and offers no protection against smallpox. It commonly persists for weeks and may last for months before it finally disappears. Associated with normal vaccination we occasionally have a general vaccinia eruption, in which the pustules may be scattered in large numbers over the surface of the body, resembling chickenpox. These pocks run through the typical stages of the local sore produced by vaccination, but they are much smaller and run their course in a shorter time. Erythematous rashes resembling German measles, mottled rose-colored patches resembling true measles, and urticaria may also occur.

Secondary Vaccinations.—Secondary vaccinations may rarely occur from the accidental transfer of the vaccine virus from the local sore to some other part of the body; this transfer usually comes through the hand of the patient. The genital organs are the most common site of secondary vaccinations. The eye may also be inoculated. Accidental vaccinia may also occur in unvaccinated children suffering from eczema. In these cases generalized vaccinia may develop and the disease may terminate fatally.

**Complications.**—The most common complication is a septic infection of the local sore by pyogenic microorganisms. As a result more or less serious cellulitis may occur, involving the tissues of the arm around the point of vaccination; even a general septicopyemia may result. Abscesses in the lymphatic glands of the axilla and neck may occur, especially in tuberculous children. Impetigo and erysipelas are rare complications. Syphilis and tuberculosis are perhaps never transmitted by vaccination. The latter disease, however, may be aggravated by vaccinia. Tetanus has been conveyed by vaccination; this, however, is a very rare occurrence, and could only happen where the animal producing the virus was suffering from tetanus.

**Revaccination.**—A successful vaccination fully protects the individual from vaccinia and from smallpox for a more or less indefinite period of time. In some instances the immunity thus produced lasts throughout a long life-time; as a rule, however, it begins to run out after five or six years, and from that time on the child becomes more and more susceptible to smallpox. During this period of partial immunity the individual may have smallpox in a mild form (varioloid). It is therefore advisable to follow the first vaccination by a second vaccination six or seven years later, and thereafter the individual should be vaccinated during every smallpox epidemic, if there has not been a successful vaccination within the last five years. Second vaccinations commonly run a mild course, and the local sore produced thereby gives little annoyance and is associated with no constitutional symptoms. An infected vaccination wound or other injuries may present a scar resembling that produced by a successful vac-

cination. The so-called typical scar should not, therefore, exempt individuals from vaccination during smallpox epidemics.

**Treatment.**—In the care of the local sore two things are sought, first, to prevent traumatic injury, and, second, to prevent infection. Immediately following the operation of vaccination the wound should be covered with clean cotton or linen cloth. This should not be bandaged about the arm, as such a dressing drags the sore in the putting on and off of clothing. A strip of clean cloth covering the sore may be held by adhesive plaster placed far enough away to not come within the zone of congestion produced by the sore. A cloth of this kind may be renewed every day and the vaccine wound dusted with boracic acid powder. If there is much itching carbolized vaselin may be used not on but around the wound. Oils, ointments and moist dressings to the sore are contraindicated as long as it remains dry and uninfected. The cloth for the protection of the wound may also be stitched to the undershirt, extending several inches above and below the point of vaccination, and this should be changed every day. This precaution is even better than changing the child's underclothing, since a soft cotton or linen rag is less irritating to the vaccine wound than the material ordinarily used for underclothing. If the sore becomes moist and adheres to the rag, dusting powders are of service; among these aristol and chemically pure boracic acid are valuable. They should be applied frequently during the day and the sore protected by a light vaccination shield, large enough not to injure it and so constructed, of wire or other material, that there will be a free access of air to the wound. If infection results in a cellulitis the patient should be confined to bed, all bandages removed and moist dressings of 1 to 1,000 bichlorid of mercury or 5 per cent. aluminium acetate, used. Vaccination shields are of special value in protecting the scab after the local inflammation has subsided.

## VARICELLA

Varicella, or chickenpox, is an acute infectious disease characterized by a vesicular eruption which is commonly preceded by fever and other slight constitutional symptoms. In its early history it was confounded with mild forms of smallpox, with which it is now known to have no connection.

**Etiology.**—Although the specific microorganism of this disease has not been discovered, it is known to be very contagious; only slightly secondary to measles in this regard. It occurs in mild epidemics, spreading rapidly through schools, families and institutions. The facility with which it spreads among the susceptible members of confined communities shows that its contagious principle is readily carried by fomites through the air to all parts of the room, and that personal contact of the sick with the well is almost always followed by transmission of the contagion. That the disease is not readily conveyed from house to house and from institution to institution is evidence that the contagion is short-lived and is per-

haps very rarely carried by a third party or by the clothing and other belongings of the patient. There is also little doubt but that the period of great contagiousness lasts but a short time, probably only during the four or five days covered by the acute symptoms; after that there is little danger of transmitting the contagion; otherwise the disease would be widely spread by convalescent patients, still carrying the scales and scabs of the eruption. Varicella is preeminently a disease of childhood. Perhaps 90 per cent. of the cases occur in children under ten years of age. In adults and nurslings it is rare.

**Incubation.**—By most writers this period is placed at about fourteen days; it may, however, be a few days longer or shorter, and Gregory states that it is less than one week.

**Symptomatology.**—The ONSET is marked by a rise in temperature, associated with headache, nausea, and sometimes with chilly sensations. The fever preceding the eruption is slight, but later it may rise to 103° F. and in severe cases even to 105° F. It lasts from two to six days, and is rather markedly remittent. The headache, nausea and general discomfort disappear within thirty-six hours and thereafter the patient is comfortable.

FIG. 58.—VARICELLA ERUPTION ON THE FOURTH DAY. (Hecker and Trumpp.)

The EXANTHEM is the characteristic symptom upon which the diagnosis is made; it appears early and develops rapidly. In mild cases the constitutional symptoms may be so slight as to be overlooked, and attention is first called to the child's illness by the eruption. This makes its appearance first in widely scattered patches on the face and back, quickly spreading over the body and later involving the arms and legs. When the eruption is fully developed it is much more marked over the body than on the face; in well-marked cases the body may be almost covered while the face shows comparatively few vesicles. The eruption first appears as violet-pink macules, which later become small vesicles varying greatly in

size, from 1 to 10 mm., the average size being about 3 or 4 mm. These vesicles are filled with a clear fluid which later becomes cloudy; they are surrounded by a small erythematous zone. Within twenty-four or forty-eight hours they begin to dry up, and during this period of desiccation may appear slightly umbilicated; this umbilication is due to the drying process preceding scab formation. The small dark scab, which marks the site of the vesicle, may remain for two or three weeks before it finally falls off. The chickenpox eruption is characterized not only by the great variation in the size of the vesicles, but more especially by the fact that they appear in successive crops on the same parts of the body, so that where the eruption is most profuse *the hand may cover chickenpox vesicles in every stage of development,* including the tiny red macule, the matured vesicle and the desiccated scab. The skin eruption during its height is usually associated with itching; this symptom may continue for days.

The enanthem may be of value in the differential diagnosis. It appears in the form of vesicles or more frequently as an erosion of the mucous membrane locating the site of a ruptured vesicle, and is surrounded by a pinkish-red zone. Following the breaking of the vesicle the erosion is frequently covered by a thin white exudate. This eruption occurs most commonly on the soft and hard palate, but may also appear on the pillars of the pharynx, tonsils and rarely on the gums and tongue. The enanthem is coincident in appearance with the exanthem. Painful and distressing local symptoms may be produced by pocks in unusual places; in the ear they may produce earache, in the throat an irritating and harassing cough, at the mouth of the meatus urinarius painful urination, and in the vulva an uncomfortable swelling.

Blood.—There is a moderate leukocytosis during the pustular stage with an increase of polymorphonuclears; no eosinophiles.

**Complications.**—While the course of chickenpox in the vast majority of cases is very benign and the prognosis in uncomplicated cases is invariably good, it should be remembered that it may be followed by serious complications. Tuberculosis is the most common. Bright's disease, gangrenous dermatitis, adenitis and arthritis may rarely occur.

**Diagnosis.**—The only disease with which chickenpox may be confused is varioloid. From this it may be differentiated by the fact that in chickenpox every stage of the eruption may be found on the same part of the body at the same time, while in smallpox the skin lesions on any part of the body are in the same stage of development. In chickenpox also there is greater variation in the size of the vesicles and they do not have the early shotty feel of the smallpox vesicle. As emphasized by Councilman the vesicular fluid of the chickenpox vesicle is contained in a single cell and not in a system of cells as in smallpox, so that a single pin prick at once flattens the chickenpox vesicle. The vesicle in chickenpox is never, as in smallpox, umbilicated, although it may appear slightly so during the stage of desiccation. The chickenpox vesicle runs its course in three days,

the smallpox vesicle requiring a much longer time. The two diseases may also be differentiated by their characteristic enanthems.

**Immunity.**—One attack, as a rule, confers lasting immunity, second attacks are extremely rare.

**Prophylaxis.**—Patients suffering from this disease should be isolated. Tuberculous children should be especially guarded, as chickenpox may aggravate an existing tuberculosis. Because of the mildness of this infection it is practically impossible to continue the quarantine longer than one week. This, however, covers the period of greatest contagiousness.

**Treatment.**—Chickenpox is a mild, self-limited disease, which in the great majority of instances requires little or no treatment. A preliminary laxative should be given and during the acute febrile stage the patient should be confined to bed. The diet should be simple and composed largely of milk, cereals, purées of vegetables, bread and fruit juices. Albuminous foods should be avoided or partaken of sparingly, the object being, as in the other acute infections, not to overtax the kidneys. Phenacetin and antipyrin may be given to relieve the headache, reduce the fever, and make the patient more comfortable during the acute febrile stage, but these drugs should be used only when they are needed and not as a routine measure. Lanolin and carbolized vaselin may be applied to the skin to relieve the itching. Care should be taken to prevent the patient from scratching the pocks, as this may result in infection and in the production of unsightly scars.

## CHAPTER XLII

### MUMPS

### (*Epidemic Parotitis*)

Mumps is an acute infectious disease characterized by fever and by inflammation of the salivary glands, especially the parotids.

**Etiology.**—The specific microorganism is unknown. The investigations, however, of Leveran and Catrin are of importance since they found in the blood, in the parotid gland, in the testicle and in the edematous fluid diplococci which arranged themselves in twos and fours. Diplococci have also been found in Steno's duct and in abscesses of the parotid, complicating mumps. Further investigations, however, are required to show whether this organism is etiologically related to mumps. The specific cause is spread by the well coming in close contact with the sick. The poison cannot be carried any distance through the air, and is rarely transferred by a third person. Such close contact is required for the spread of this contagion that there is little difficulty in confining it by quarantine regulations. I have on many occasions quarantined cases of mumps in the end room of a children's ward without having the infection spread.

In hospitals and in other institutions where a large number of children occupy the same room the disease spreads to children in neighboring beds rather than to children across the ward. The disease occurs in epidemics, little influenced by weather conditions, but they are slightly more common in winter than in summer. The severity of the disease and the degree of its infectiousness vary greatly in different epidemics. Instances are on record where one-half of the exposed children have contracted this disease. In milder epidemics not more than 20 per cent. of those exposed contract it.

Age.—Comby reports a congenital case. The disease is almost unknown under one year and is very rare under two and over forty; susceptibility increases up to the sixth year. The great majority of the cases occur between the sixth and fourteenth year of life. After this susceptibility gradually diminishes, young adults about twenty years of age still being quite susceptible, but in old age the disease is almost unknown.

**Period of Contagion.**—This lasts for three weeks dating from the beginning of the attack. The disease, however, may be contagious for a few days before the acute symptoms have developed, and in some instances it appears that the contagion may last for five or six weeks. It may be assumed that the period of greatest contagion is during the first week while the acute symptoms are present, and that it gradually diminishes during the next two weeks.

**Incubation.**—Eighteen days is the average period of incubation, but it may vary from two to four weeks.

**Immunity.**—One attack commonly confers immunity. Second attacks are unusual and third attacks rare.

**Symptomatology.**—The child is fretful, languid, sleepless, loses its appetite, has a slight elevation of temperature with headache, backache, and a certain amount of stiffness and tenderness at the angle of the jaw. Shortly after the onset of these symptoms, usually in from one to three days, the swelling of the parotid gland appears, and there is localized tenderness and an increasing stiffness in the movement of the jaw. The swelling appears between the angle of the lower jaw and the mastoid process. It gradually obliterates the intervening depression, rises and extends in front of the ear, involving the whole gland. The subcutaneous tissue surrounding the parotid not infrequently becomes infiltrated and edematous. This tumor mass, which is the characteristic sign of the disease, is tense and firm, does not pit on pressure, is located in front of the ear and extends downward, tilting the ear backward and extending into the neck. In some instances only one parotid is affected, but, as a rule, the other begins to swell three or four days later, so that in most instances the disease is bilateral, giving a peculiar squirrel-like appearance to the face; the neck, just under the ears, is wider than the face itself.

The sub-maxillary and sub-lingual glands may also be involved, producing firm, resistant, tender, swollen masses. These glands may be affected after or before the parotids, or the disease may be wholly confined

to the sub-maxillary and sub-lingual glands. The swelling in the parotid gland gradually reaches its height in four or five days, remains about the same for two days and then rather quickly subsides, lasting in all from one week to ten days; in the bilateral cases this period may be slightly prolonged if one parotid is infected some days after the other.

There is more or less pain and tenderness associated with the parotid swelling which may be aggravated by the taking of acids or spicy foods which may irritate the buccal mucous membranes. The pain is increased on opening the jaw. This causes the patient to keep his mouth partly closed in speaking and in taking food, and in unilateral cases to tilt the head slightly to the diseased side so as to relax the tension of the muscles. Sore throat is commonly complained of on swallowing and an examination of the mouth shows a swelling and congestion of the buccal mucous membrane around Steno's duct. The tonsils, soft palate and fauces are red and congested.

The fever, which is one of the earliest symptoms, rises by the second day to about 102°F. In severe cases it may reach 104°F. This continues for four or five days and falls to normal soon after the parotid swelling reaches its height. The fall in temperature may be postponed or interrupted by the involvement of other glands or by relapses which may occur during the period of apparent convalescence.

Epistaxis may be a feature of the disease in certain epidemics. Slight enlargement of the spleen and external lymphatics may occur in severe cases. There may be a slight lymphocytosis in the early stages, and if orchitis occurs leukocytosis may be marked.

The duration of mumps in uncomplicated cases varies from one to two weeks. Severe cases may last much longer, and mild afebrile cases may show acute symptoms for only a few days.

**Complications.**—In childhood this disease runs a much milder course than it does in adults, and the complications so common and so much dreaded in the adult are rarely seen. For this reason excellent authorities have advised that children should not be quarantined from this disease, as it is much better for them to have it in childhood than to run the risk of having it in a more severe form in adult life.

Orchitis, the most common and dreaded complication in adult life, sometimes occurs in boys between the ages of twelve and fourteen; it rarely occurs earlier. This complication usually appears about the end of the first week of the disease. The testicle is very tender and may be swollen to two or three times its normal size. In the female ovaritis may occur. In both sexes there may be enlargement and tenderness of the breasts. Deafness is rare, but it may result from a complicating otitis media. Albuminuria is not infrequent, but nephritis is very rare in childhood; it occurs more frequently in adults. Suppuration of the parotid, paralysis of the facial and auditory nerves, pancreatitis and inflammation of the lacrimal gland are almost unknown in childhood.

**Diagnosis.**—Mumps must be differentiated from other forms of paro-

titis, and the physician should keep in mind the fact that inflammation of this gland from other causes is not very uncommon. It may occur as a complication of any of the acute infectious diseases, it may be a part of a general septic process, it may be secondary to stomatitis and catarrhal inflammation of Steno's duct. If these facts are kept in mind the differential diagnosis of mumps can readily be made.

**Prognosis.**—This is almost invariably good. In older children complications may leave more or less serious results, such as deafness and impotency.

**Prophylaxis.**—It is difficult to isolate these cases except during the acute stage, but for a period of three weeks from the beginning of the disease they should not be allowed to return to school, go to children's parties, or otherwise come into close contact with other children.

**Treatment.**—Mild cases require no treatment beyond confinement to the house for a few days. In the more severe cases the patient should be kept in bed for a period of eight or ten days covering the acute symptoms. During this time the mouth should be carefully syringed or washed out several times a day with a mild alkaline antiseptic. The parotid swelling should be treated with hot applications, which help to relieve the pain and discomfort. Ointments containing glycerin, belladonna and guaiacol are also recommended. Phenacetin or antipyrin with small doses of tincture of strophanthus are of value in relieving the pain and restlessness. Chloral hydrate is a valuable hypnotic in this disease, and should be given in proper doses at bedtime, and repeated, if necessary, at three or four-hour intervals to produce sleep.

"It has been shown that urotropin is excreted through Steno's duct. On this basis it should be of value in the treatment of mumps. A series of cases of mumps in adults at the Cincinnati Hospital was treated with urotropin; none of these developed orchitis, although many of a control series treated without urotropin did. The urotropin cases ran a uniformly milder course than the others." (A. Friedlander.)

Dietetic Treatment.—Acids and foods which irritate the mucous membrane of the mouth increase the pain and discomfort and should therefore be avoided. Since the patient may have difficulty in opening his mouth and cannot properly masticate his food, the diet should consist of milk, gruels, cereals, ice-cream, eggs, milk-toast and other soft and liquid foods.

## CHAPTER XLIII

### SYPHILIS

Syphilis is an acute infectious disease, due to the spirochæta pallida. It may be acquired by direct contact or it may be congenital. The acquired form of the disease, so common in the adult, is comparatively rare in the child. The congenital form, which is the ordinary syphilis of infancy and

childhood, is characterized by cutaneous eruptions, by general malnutrition and by destructive lesions of bones and internal organs.

**Etiology.**—The specific cause of this disease is the spirochæta pallida, first described by Schaudinn in 1905. This organism has been found in the various lesions of both the acquired and congenital forms of this disease, such as the initial sore, the mucous patches, the lymph glands, the skin lesions and syphilis of the internal organs.

Acquired Syphilis.—Acquired syphilis in infancy and childhood presents the same clinical picture that it does in the adult. The initial lesion, which is followed by secondary and tertiary symptoms, is contracted by direct contact with the contagion. Denuded surfaces of skin or mucous membrane on the infant or child are inoculated with the specific microorganism of this disease by coming in contact with the primary sore or mucous patches of an infected individual. In older children it may be communicated by sexual contact. Acquired syphilis, however, because of its comparative rarity and the sameness of the clinical picture which it presents to that of syphilis in the adult, requires no further consideration.

Congenital Syphilis.—Congenital syphilis is essentially a disease of infancy and childhood, although its manifestations may continue in a modified form throughout the life of the individual. The clinical picture of this form differs essentially from the acquired form and requires, therefore, careful clinical study.

Congenital syphilis can be transmitted to the child only through the mother, and the virulence of the infection and the degree of syphilization of the fetus depend upon the virulence and stage of the disease in the mother. The most virulent infections occur at the height of the maternal secondary stage; as this stage passes into the tertiary, the infection of the fetus becomes less virulent and less certain. In the very early primary and the well-advanced tertiary stages the syphilitic taint may not be sufficient to produce syphilis in the infant. Previous treatment of the maternal parent may so materially modify the infection of the offspring that it may show no signs of the disease, but the recurrence of symptoms and relapses following intermittent treatment in the mother may be followed by evident syphilis in subsequent children.

For many years it was very generally believed that syphilis could be transmitted to the child from either father or mother or from both. It was thought that the father could infect the ovum directly with his syphilized sperm and the mother escape subsequent infection through the placental barrier. It was frequently observed that these children with their mucous plaques could suckle at the breast of their mother with impunity, and the mother escape every sign of infection; a wet nurse, however, not previously infected with syphilis might be infected by these infants. It was therefore believed that the mother escaped infection and became immunized against syphilis through her syphilitic offspring (Colles' law). It was also observed that a healthily conceived child whose mother became infected at some period during parturition could be born

healthy and escape infection through the same placental barrier, and would remain immune against syphilis from maternal and other sources (Profeta's law).

The Wassermann reaction has demonstrated that both infant and mother are syphilized under these conditions. The circulation of both mother and child is extremely intimate, the placenta presenting no barrier against the infection; a mother who produces a syphilitic child must of necessity share the infection, and her immunity and that of her child is apparent, not real. If the infected mother is suffering from severe manifestations in the secondary stage, the disease would probably be transmitted in a severe form, resulting either in the death of the fetus or in advanced congenital syphilis.

Syphilis cannot be transmitted to the third generation. The physical defects, however, resulting in a weak progeny may be transmitted, but the specific lesions of this disease do not pass to the third generation.

**Pathology.**—Post-mortem examination of a macerated syphilitic fetal corpse not uncommonly fails to show any characteristic anatomical changes of this disease. This is especially true if death occurs before the fourth month of fetal life. After the fourth month the characteristic lesions commence to make their appearance, osteochondritis and enlargement of the spleen, liver, kidneys, pancreas, thymus, and indurative changes in the lungs occur with increasing frequency. Skin lesions are not commonly found until near the end of the normal period of uterogestation.

Syphilitic osteochondritis is the earliest, most common and most characteristic lesion of congenital syphilis. It occurs principally in the long bones at the junction of the shaft with the epiphysis, and the inflammation may result in the dissolution of this junction, thus separating the epiphysis. The enlargement and induration of the spleen, liver, kidneys, lungs and pancreas, so commonly present, are due to a general round-celled infiltration and connective tissue proliferation.

**Symptomatology.**—Syphilis is one of the most common causes of repeated abortions. Following an ABORTION, which may occur as early as the third or fourth month, producing a dead and macerated fetus showing no distinctive syphilitic lesions, the same woman impregnated by the same man may, one or two years later, in the seventh or eighth month of uterogestation give birth to a dead infant, showing an osteochondritis of the long bones, enlargement of the spleen and liver, fatty degeneration of the placenta, round-celled perivascular infiltration of the umbilical cord and other characteristic signs of congenital syphilis. One or two years later the same mother may give birth to a still-born infant at term or to one that lives but a few days. In this infant, in addition to the signs just noted, the kidneys, lungs, pancreas and other internal organs may show characteristic syphilitic lesions and the skin may be covered with a large bullous eruption known as syphilitic pemphigus. Still later this mother may give birth to an infant apparently normal at birth which, within the first three months, develops a syphilitic coryza followed by other signs

of congenital syphilis, and later in her life she may give birth to an apparently normal child which never shows any sign of syphilis. This chain of clinical manifestations illustrates the fact that the power of transmitting the syphilitic poison is gradually lost by the parents, and also that in direct proportion to the potency of the poison in the parents, the earlier and the more virulent will the manifestations be in the fetus, thus producing successively in the same mother abortions, premature births, still-births at term, syphilitic weaklings that live but a few days, apparently normal infants that later develop syphilis, and finally children in whom no signs of syphilis ever develop.

From what has been said it is evident that the earlier the manifestations occur after birth the more severe the disease will be. Even in very early syphilis the symptoms are not, as a rule, present at birth, but in quite a large percentage of the cases the disease manifests itself during the first or second week. In these early cases the infant is profoundly malnourished, and as the disease progresses it becomes more and more wasted, its dried skin hanging in folds, its wizened face having an aged appearance, and a well-marked coryza discharging an irritating fluid excoriates the upper lip. The lips are cracked, the corners of the mouth fissured, and mucous patches may be found in the mouth and in the anus. A bullous eruption appears on the palms of the hands and the soles of the feet. Tenderness and swelling may also be present at the ends of the long bones near the joints of the arms and legs. External and internal hemorrhages may occur from mucous membranes. In the worst cases, as the disease progresses, the emaciation becomes more extreme and the child dies within a few weeks. Fortunately, however, in the great majority of cases the clinical manifestations do not develop until after the middle of the second week of life, and from this time until the end of the sixth week is the most common period of onset; it very rarely develops after the third month. These cases are not so violent in their clinical manifestations and are much more amenable to treatment.

Coryza, or rhinitis, is the commonest and most characteristic symptom, and, as a rule, marks the onset of the symptom-complex. The mucous membrane of the nose is intensely irritated, swollen, and discharges a mucopurulent fluid, which may be tinged with blood. This discharge is irritating in character, producing an eczema of the upper lip. Crusts form in the nasal cavity, which have a tendency to retain the discharge, and this retained discharge, as the air passes through it, produces a snuffling sound which has been characterized as the "snuffles." With the formation of crusts and the retention of discharges the mucous membrane of the nose becomes more or less disintegrated, and the cartilage and bones of the nose become involved. If this process continues marked nasal deformities may result. The nasal septum may be perforated and the bridge of the nose may be broken down, producing the saddle nose and other deformities. A severe coryza so obstructs the nasal passages as to materially

interfere with the infant's nursing. A coexisting laryngitis may produce hoarseness; this is a very suggestive symptom.

Skin Lesions.—Vesicular eruptions are very rare in hereditary syphilis, except the large vesicular or bullous eruption known as syphilitic pemphigus, which occurs in the severe forms of this disease found during the first weeks of life. This eruption may occur over the body, but is commonly confined to the palms of the hands and the soles of the feet, and is composed of large blebs from an eighth to one-half inch in diameter, filled with bloody fluid, and the intervening skin where the eruption is profuse is indurated and dark red in color; where the blebs are isolated a zone of such tissue surrounds them. This eruption not uncommonly causes complete exfoliation of the skin on the palms of the hands and the soles of the feet and is most ominous in its significance, as it occurs only in the worst cases. It is not to be confounded, however, with non-syphilitic pemphigus neonatorum, due to infection, and which may occur in well-nourished infants. This eruption is not surrounded by a reddish-brown base, and does not select the palms and soles as its favorite site. Hochsinger has described another condition of the skin which is characteristic of hereditary syphilis and does not belong to the acquired form. The skin is diffusely infiltrated, thickened and feels dense and firm to the touch, has a dark red, shiny appearance, getting darker in color as it gets older. This diffused infiltration of the skin is commonly associated with other eruptions, very like certain of the eruptions that are found in the acquired form of syphilis. These eruptions, like the pemphigus eruption previously described, may be superimposed upon this dark red indurated skin. Among them the maculopapular syphilide is the most common. This occurs as small, round, rose-red spots which, as they grow older, become more or less copper-colored, and are elevated above the surface of the skin. A distinctly papular eruption may be associated with this macular eruption, the small papules marking the center of the rose-colored macules, or appearing as a separate exanthem. A pustular or a papulopustular syphilide may also appear. The pustular eruption occurs most commonly on the face, thighs and buttocks. Mixed eruptions are very common in hereditary syphilis, and the macular, papular and pustular syphilide may all be present at the same time.

Infiltration of the skin of the face may give it a tense, glittering appearance, and the same condition may exist about the region of the anus. This leads to cracking of the skin and the formation of *radial fissures* about the corners of the mouth and the anus, which produce a more or less characteristic appearance and one of the most valuable of diagnostic signs. Ulcers and mucous patches may develop in these fissures, greatly increasing the irritation, and papular excrescences may develop about the rectum, producing small tumors called condylomata. The infiltration of the skin about the finger nails produces paronychia. This may be an active ulcerative process about the root of the nail, or it may be a lower grade of inflammation unaccompanied by purulent discharges.

The nail may be destroyed or distorted. This same process affecting the hairy parts, such as the eyebrows and the scalp, may result in the complete destruction of hair in these regions. Complete baldness, however, is not very common, especially in early infancy.

Bones.—The long bones may be tender and enlarged near their extremities, at the junction of the shaft and the epiphysis. These sensitive swellings occur with special frequency at the lower epiphyses of the humerus and femur, and separation of the epiphyses may result, as shown by increased motion and crepitus. Associated with these symptoms there may be almost entire loss of motion of the affected limb. This is spoken of as syphilitic pseudo-paralysis, and is, according to Hochsinger, purely a muscular manifestation, due to severe periosteal involvement at the point of muscular attachment. According to the same authority syphilitic phalangitis primarily involves the first phalanges of either the fingers or the toes. The fingers are most commonly affected and the index finger is its favorite site. Following the involvement of the proximal phalanx, the middle phalanx of the same finger or toe may be involved. The swelling thus produced is chronic in character, conical in shape, painless, tense, and glossy. The soft parts are but slightly involved, and ulceration rarely occurs.

Fig. 59.—Syphilitic Dactylitis; Infant Sixteen Weeks Old. (Max Dreyfoos.)

The teeth are delayed, imperfectly developed and decay early. The skull presents more or less characteristic rachitic changes with its open fontanels and enlargement of frontal and parietal eminences, and craniotabes may occur.

General Malnutrition.—The degree of malnutrition will depend largely upon the severity of the disease and the food of the infant. In the severe cases developing soon after birth malnutrition is profound and, as previously noted, commonly progresses to a fatal termination. In the later and less severe cases the child at birth may be fairly well nourished and, if it have the advantage of good breast milk and early and proper treatment, it may continue in a fair state of nutrition. Syphilitic babies, however, as a rule, have feeble digestive capacity, and those that are artificially fed show a more or less marked malnutrition, which may,

even in the cases that develop some weeks after birth, become very pronounced. Malnutrition is associated with more or less marked anemia, which may be characterized in severe cases by a diminution in the amount of hemoglobin and in the number of red blood corpuscles. Many of the red blood corpuscles are nucleated and vary greatly in size, microcytes and megalocytes being present. There is also a more or less marked leukocytosis with a preponderance of myelocytes. Eosinophiles are also present. General lymph-node enlargement does not, as a rule, occur in early hereditary syphilis.

Brain.—Disease of the brain and its membranes may produce hydrocephalus, idiocy and hemiplegia.

Spleen.—This organ is very much enlarged and is easily palpated in the syphilis of early infancy. The earlier the disease develops the more pronounced is this sign.

Liver.—The liver may be enlarged, extending well below the margin of the ribs; jaundice may occur.

Kidneys.—Acute nephritis may occur early in the disease and yield to specific treatment. It is also a late manifestation, occurring shortly before death. In these cases the nephritis may be a terminal lesion, resulting from the intestinal and general toxemia.

Late Hereditary Syphilis.—Late hereditary syphilis develops later in the life of the child, usually after the fifth and sometimes as late as the twelfth or fifteenth year. These cases present the symptoms of ordinary tertiary syphilis. They are commonly believed to be true hereditary syphilis, the symptoms of which, for some unexplained reason, were not clearly manifested in infancy. The small minority of the cases, however, may be due to an overlooked syphilis acquired earlier in life. In calling attention to these cases Hutchinson described the following triad of symptoms which are more or less characteristic: First, the notching of the central incisor teeth; second, an interstitial keratitis; and third, sudden deafness without apparent local cause.

*Hutchinson's Teeth.*—The central incisors of the secondary teeth have a large, single, crescent-shaped notch occupying the center of each tooth. The teeth themselves are rounded and taper from a broad base to a constricted cutting edge, presenting a peg-like appearance. They are inclined toward each other, as a rule, but occasionally they may diverge. When present, although not absolutely pathognomonic of syphilis, they are, when taken in connection with other symptoms, among the most valuable signs of this disease. They are absent, however, in a majority of the cases.

Fig. 60.—Hutchinson's Teeth.

*Interstitial keratitis* may be associated with corneal opacities and with inflammation of the iris, but, as a rule, is not accompanied by an active conjunctivitis. This symptom is also especially valuable in its association with other symptoms of hereditary syphilis.

Sudden *loss of hearing* unaccompanied by apparent disease of the ear is very suggestive of hereditary syphilis. Loss of hearing and mastoiditis may also occur in this disease, resulting from a low grade of chronic otitis.

*Periostitis* of the tibia, ulna, radius, and humerus may occur. The tibia is most commonly affected, and as a result a long, narrow, tender swelling is presented on its anterior surface.

The *nose, pharynx* and *palate* may be involved in destructive ulcerations, causing necrosis of the underlying bones, and marked deformities may result. In severe cases the ulceration in the nasopharynx may produce widespread destruction of the tissues, causing the bridge of the nose to give way, forming the so-called saddle nose.

*Gummatous ulceration* may occur over the *skin,* especially of the face and legs, producing large round ulcers with indurated borders. These ulcers have a tendency to group themselves and in their healing produce large radiating scars which are more or less characteristic. This is especially true when these ulcers involve the mucous membrane of the lips. Hochsinger says: "An absolutely positive proof of former hereditary syphilis is found in the radial scar formation of the lips."

The *spleen* is almost always notably enlarged, more so than any of the other internal organs; the liver may be increased in size. There is a more or less notable enlargement of the external lymph nodes; in this particular late hereditary syphilis differs from the infantile form.

*General malnutrition* and retarded and perverted development are among the notable symptoms occurring in late childhood about the period of puberty.

**Diagnosis.**—The chief difficulties in the differential diagnosis of early syphilis are presented by infantile marasmus and tuberculosis. In infantile marasmus the malnutrition commonly occurs later in the life of the child, and there is perhaps no history of previous abortions and snuffles. Enlargement of the spleen and characteristic syphilitic skin eruptions are absent, while on the other hand there is perhaps a history of gastrointestinal disease or other causes which may explain the marasmus.

Tuberculosis of the bones of the finger and of the long bones of the arm and of the leg may be mistaken for syphilis. The differential diagnosis may here commonly be made by the family and personal history of the child, supplemented by the tuberculin skin test. Tuberculous dactylitis involves not only the bone, but the soft parts as well, producing a tender and more or less acute inflammation which tends to suppuration and ulceration of the soft parts. In these particulars it differs from the syphilitic dactylitis previously described. In the long bones tuberculosis involves the epiphyses rather than the shaft of the bones, and the resulting

inflammation affects the joints, while in syphilis the joints are not commonly involved and the diaphyses of the long bones are the sites of the inflammation.

The Wassermann-Neisser-Bruck reaction for syphilis is the most exact method of differential diagnosis. To make this test accurately, however, requires special training and laboratory equipment. A detailed description of its technique would, therefore, be out of place here, but a brief allusion to its fundamental principles and their practical application will not be amiss.

Substances called antibodies are formed in the serum of every syphilitic who in any way reacts against the infection. They are constantly present in the vast majority of the untreated or inadequately treated cases of syphilis in all stages of the disease, and for a period of years. These substances preclude hemocytolysis or the solution of the sheep's corpuscles in the Wassermann reaction. This is a specific reaction for syphilis, and when positive is an unfailing evidence of the presence of the disease. It is frequently absent in well-defined cases of severe or malignant syphilis, and in such cases there is evidence of the failure of the system to properly react against the ravages of the affection. It gradually disappears in cases that have received adequate medication, and in such cases it becomes the best scientific evidence we possess that the disease is under control. Hereditary syphilis, according to Ledermann and numerous others, will often show a positive Wassermann reaction into early adult life or later.

In view of the now generally accepted fact that a syphilitic infant is of necessity progeniated from a syphilized mother with active syphilis, a Wassermann examination of the mother confirms or rules out the diagnosis of syphilis in a suspected infant. This has a practical application inasmuch as the blood for the examination can be more easily obtained from the mother. The blood may be secured by allowing it to flow through an 18-gage needle into a sterilized centrifuge tube from one of the large veins of the forearm, after an Esmarch bandage has been placed above the elbow. In an infant the blood is most conveniently collected under less favorable and less aseptic conditions by a scarification below the scapula.

**Prognosis.**—The earlier the symptoms appear the worse the prognosis. The more severe cases, and this includes a large percentage of the total number, die *in utero,* and of those that are born alive the prognosis is almost uniformly bad when the symptoms make their appearance during the first week. These two classes include in the neighborhood of 50 per cent. of all cases. In the milder cases, in which the symptoms appear after the second week, the prognosis under proper treatment is good so far as life is concerned. The vast majority of these respond quickly to the specific medication of this disease, and their subsequent chances for life will depend largely upon their hygienic surroundings and their dietetic treatment. Relapses occur in a large percentage of these cases, because of insufficient treatment. It is also probable that hereditary syphilis usually results in more or less permanent physical deterioration. The degree,

however, of this deterioration can be very materially modified by early and persistent treatment.

**Prophylaxis.**—Individuals affected with syphilis should not marry for four years after the beginning of the disease, and then only after at least two years of well-directed medical treatment. If married, conception should be prevented during the two or three years necessary to control this disease. If, however, conception occurs, the mother should have antisyphilitic treatment throughout the entire period of pregnancy. In this way it is possible to largely protect the fetus and cause the mother to give birth to an apparently healthy child, which later may or may not show signs of this disease. Special stress should be laid upon the value of giving the mother antisyphilitic treatment in all cases where this disease is suspected. This is especially important if the mother at any time during her life has ever been actively syphilitic. With the birth of a syphilitic infant the nurse or parents should be impressed with the fact that the infant has an infectious disease which may be communicated to others. Children are in special danger from kissing and from using the same food utensils. The danger also of infection to a non-syphilitic wet-nurse from mucous patches in the mouth of the infant should be explained. The danger from the contagion of hereditary syphilis has no doubt been greatly exaggerated, because of the dread of this disease. But although this danger may be slight, there is no reason why every possible precaution should not be taken to prevent the infant from contaminating others.

**Treatment.**—Dietetic Treatment.—The dietetic treatment of infantile syphilis is most important. Since these infants have feeble digestive capacities and at the same time have a more or less marked malnutrition to overcome, it is all-important that they should, if possible, be given breast milk. Fortunate it is, therefore, for the syphilitic infant that its mother may nurse it with comparatively little danger to herself. The mother's milk, therefore, should be used in every instance where it is possible, and when insufficient should be supplemented by modified milk formulas suitable to the age and digestive capacity of the infant, and these supplementary feedings should be carried out under the principles outlined under Mixed Feeding. That is to say, the infant is to be given the breast milk at every nursing, and this is to be supplemented, if necessary, by the bottle. In those cases where mother's milk is not available it may sometimes be necessary in order to save the life of the infant to employ a non-syphilitic wet-nurse, having her bring her own infant with her and giving the syphilitic infant such breast milk as can be obtained by pumping from the breast of the wet-nurse. Under no conditions, however, even though the mouth of the syphilitic infant be apparently normal, should it be allowed to nurse the milk directly from the breast of the non-syphilitic wet-nurse. Great importance is here laid upon the value of breast milk in the treatment of this disease, because I believe that it is necessary to complete success in the treatment of the great majority of these cases. When, however, the breast milk is not available the infant is

to be fed according to the rules laid down for weaklings under Chronic Intestinal Indigestion.

Medical Treatment.—*Mercury.*—Mercury is a specific for this disease, so much so that a symptom group in an infant over three weeks of age that fails to respond to this treatment is not syphilitic. This therapeutic test is therefore a diagnostic measure of great importance.

Inunction of Mercury.—In the young infant mercury may perhaps be given more satisfactorily by inunction than by any other method. For this purpose unguentum hydrargyri mixed with anhydrous lanolin should be used. A quantity of this ointment sufficient to represent ten or fifteen grains of the mercurial ointment is to be used for each inunction. The site of the inunction should be prepared by carefully cleansing with warm soap and water and then, after thoroughly drying the skin, the mercurial ointment is to be gently rubbed in for from five to eight minutes, the operator using rubber gloves in making the inunction. In beginning the routine treatment one application is made daily, and the sites commonly selected are the inner surfaces of the thighs, the sides of the chest beneath the axilla, the lower abdomen and, if necessary, the flexor surfaces of the lower arms and legs. It is better to rotate in using these various sites for inunctions, as the continuous application of the ointment to the same portion of the body day after day may produce an irritation of the skin. In the average infant the specific therapeutic action of mercury can be more quickly and more satisfactorily obtained by inunctions than by any other method, and its administration in this way is accompanied by no gastrointestinal disturbance. These facts should make the inunction method the method of election. It should be remembered, however, that satisfactory results can be obtained only by carefully following the technique as above outlined. The mercurial ointment should be diluted with lanolin and should be thoroughly rubbed *into a clean, dry skin.* Following this application the child may have a bath and the unsightly ointment removed from the skin. This is sometimes necessary in the treatment of these cases in private families where it may be important that the nature of the treatment should not be known to all the members of the household.

Internal Administration of Mercury.—The administration of mercury by the mouth is by far the most popular method. The vast majority of cases in private practice are treated in this way, because almost as good results can be obtained by this method and because it is so easy to give mercury in this manner. For this purpose four preparations are in common use, mercury with chalk, calomel, bichlorid of mercury and protoiodid of mercury. Of these mercury with chalk is the favorite with the majority of English and American pediatricians. It may be given in from one-half to one-grain doses two or three times a day to young infants. The size of the dose may vary with the results obtained and with the condition of the gastrointestinal canal. If small doses are being given (½ grain) and the symptom group does not yield readily to treatment, the doses are to be gradually increased to 1 or 1½ grains, and in older children to 2

or 2½ grains. When the symptom group has been controlled it is better to return to the smaller doses for the long-continued treatment of the disease. This rule applies in the administration of all of the mercury preparations whether they be given by the mouth or by inunction. The larger dose that is necessary for the quick control of the early symptoms, is from one-third to one-half too large for the continued administration of this drug over long periods of time. The only advantage that the mercury with chalk has over the bichlorid and protoiodid is that it commonly produces less gastrointestinal disturbance and is therefore more suitable for long-continued use. This applies only to infants.

The bichlorid of mercury may be given to infants in from 1/150 to 1/200 of a grain, well diluted, two or three times a day. The total quantity administered in twenty-four hours should vary from 1/100 to 1/40 of a grain. The larger dose perhaps being required to control the acute symptoms, and the smaller dose to be used later for continuous administration. The protoiodid of mercury is used very largely by the German school of pediatricians, who believe that better results are obtained from this preparation than from any other. It may be given in doses of 1/60 of a grain three times a day.

Calomel is a remedy of great value in beginning the treatment of hereditary syphilis, and it is asserted by some authorities that the initial specific action of mercury can be obtained more quickly with calomel than with any other preparation of this drug. It may be given in 1/10-grain doses at three or four-hour intervals.

Comparative Value of the Various Mercurial Preparations.—In beginning the treatment it is perhaps advisable to commence with calomel in 1/10-grain doses at three-hour intervals. This medication may be continued for four or five days, or until decided laxative action has been produced. The calomel should then be discontinued and followed by mercurial inunctions for weeks and possibly for months, until the syphilitic symptoms are under control and the nutritional problems have been largely solved. Then mercury with chalk may be used for the long-continued mercurial course, which is to extend with interruptions over a number of years, but during this time, if gastrointestinal disturbances develop and the infant's nutrition is thereby threatened, inunctions are again to take the place of mercury by the mouth. In older infants and children the bichlorid or protoiodid of mercury may be substituted for the chalk mercury and the mercurial ointment.

*Iodin.*—Iodin is also of great value in the treatment of hereditary syphilis. It may be given in the form of iodid of potassium, iodid of sodium and iodonucleoids. The iodonucleoids will not disturb the stomach and are therefore of special value in young infants. It may be combined with equal parts of saccharated pepsin or milk sugar and given in 1 or 2-grain doses three times a day; this may be increased 1 grain for each year of life up to four years. Iodid of potassium dissolved in milk or essence of pepsin may be administered to infants in 2-grain doses three times a

day. In children five or six years of age this dose may be increased to 5 or 10 grains. The iodid of potash is on the whole the best form for administering iodin in these cases. But if it should cause gastric disturbance iodonucleoids may be substituted. The iodids are especially indicated in late hereditary syphilis, but they may also be indispensable in the treatment of infantile syphilis when gummatous ulcerations and bone lesions are present.

*Duration of Treatment.*—It has for many years been my practice to give mercury with short interruptions during the first year, and for about half the time during the second year. After the second year, as a matter of routine, a course of iodids should be occasionally administered to alternate with a course of mercury, and this should be continued until the child is four or five years of age. These courses should be of six weeks' duration and should be given only two or three times during the year. After the fifth year the treatment should be resumed on the appearance of chronic obscure symptoms of any kind. If at any time pronounced and active symptoms of syphilis reappear, vigorous and prolonged antisyphilitic treatment must again be instituted. The long-continued interrupted use of mercury and the iodids produces no bad results on the teeth or other developing structures.

*Salvarsan.*—Salvarsan has proved very efficacious in the treatment of congenital syphilis. The mortality of this disease in the new-born has been materially reduced by the introduction of this remedy. Under it the gain in weight and marked improvement in general appearance of the infant have been as noteworthy as the rapid disappearance of active symptoms. The administration of this remedy to infants is attended with technical difficulties. The intravenous and deep muscular injections with alkaline solutions are impracticable. The freshly precipitated, carefully neutralized salt of salvarsan must be injected subcutaneously. These injections are fairly well tolerated, although they produce painful infiltrations and occasionally form abscesses and indolent ulcerations of the overlying skin.

Six decigrams of salvarsan, under careful aseptic precautions, are dissolved in a clean mortar with thirty-five drops of sterile 10 per cent. sodium hydroxid solution; this is then diluted up to 12 c. c. with normal salt solution. Then two to six grams varying with the age, weight and development of the child (one to ten years, one to three decigrams of salvarsan) are drawn off with a sterile pipette into a second sterile mortar. A few drops of concentrated glacial acetic acid are then added with gentle stirring until the color is changed from red to light yellow, and neutrality is established; this can be determined by means of sterile litmus paper. It is then diluted to about 10 c. c. with sterile normal salt solution, transferred to a centrifuge tube, and centrifuged for five minutes. The clear supernatant liquid is then carefully drained away and the residue, after it is again diluted with 5 to 10 c. c. of sterile normal salt solution, is transferred to a syringe and injected subcutaneously under the scapula.

In view of the fact that the administration of salvarsan is occasionally attended with relapses, it is advisable, in the absence of careful Wassermann control, to supplement the administration of this remedy with intermittent treatment along the lines of the older accepted methods. It is also advisable not to repeat the salvarsan under a period of at least three or four months.

Local Treatment.—Local treatment of syphilitic ulcerations demands the careful cleansing with antiseptic washes and the use of a dusting powder composed of equal parts of calomel, subnitrate of bismuth and oxid of zinc.

## CHAPTER XLIV

### TUBERCULOSIS

**Etiology.**—Tuberculosis is a contagious disease caused by the bacillus tuberculosis of Koch. It may be general, but is commonly more or less localized, there being one or more foci of infection. It has a great predilection in childhood for the *lymph nodes,* bones, and serous membranes, but no part of the body is exempt from attack.

There are a number of fairly distinct types of tubercle bacilli; of these we are especially interested in the human and bovine, since they may produce tuberculosis in man. The human type is the most common cause of all forms of tuberculosis in the child as it is in the adult, but bovine tuberculosis is relatively much more common in children, and is not an infrequent cause of this disease in the cervical and abdominal lymph nodes and in the peritoneum. The human type is the usual cause of the more virulent forms of tuberculosis, while the bovine type, as a rule, produces a milder form of this disease.

Contagion.—Contagion is the all-important factor in the spread of tuberculosis. Tubercle bacilli are discharged from the body of a tuberculous individual in a moist state, in the sputum, the milk, the feces, the urine, and in the purulent discharges from tuberculous abscesses and ulcerations. Of these various discharges the *sputum* is by long odds the most important agent in spreading the infection, and for this reason the pulmonary form of tuberculosis is the chief source of contagion. The danger from tuberculous sputum is very great in both the moist and the dried state. The dried bacilli are much more widely disseminated than the moist bacilli, but the latter are much more active and virulent and a smaller dosage is therefore required to set up an active tuberculosis. In their moist state the tubercle bacilli are thrown in a fine spray for a distance of eight or ten feet by coughing, and may thus be inhaled by those who come within the range of this infection. In this manner and also by the careless disposal of the expectoration, the clothing and surroundings of the patient may become infected; handkerchiefs, wearing apparel, carpets, hangings and bed-clothing may be carriers for a short time of the bacilli in a moist

state, and therefore a source of great danger, especially to infants and young children who spend a great portion of the time in such contaminated apartments. Their milk and other food, which is too often prepared in such surroundings, may become contaminated, and act as a vehicle for carrying the tubercle bacilli into their intestinal canals. Another danger from the moist sputum lies in the fact that flies and other insects may be a means of transferring bacilli to remote parts of the house and producing food contamination. Infants with the inherent instinct which they have of putting everything into their mouths are in special danger, since their toys and other foreign bodies with which they come in contact may carry into their mouths the moist tubercle bacilli.

But after all the greatest danger in the spread of tuberculosis lies in the fact that the slender, rod-shaped bacillus of this disease is small and light enough to be carried short distances in a *dried state* by dust, or other foreign particles put in motion by currents of air, and thus be inhaled or produce food contamination. This in fact is an ever-present danger in public conveyances and in buildings now housing or that have housed tuberculous patients.

Next to sputum, *milk* contaminated with tubercle bacilli is the most potent factor in the spread of this disease. There is no longer any doubt but that milk from tuberculous cattle may be a source of danger, especially to infants, and the milk of the tuberculous mother may also be a carrier of tubercle bacilli. But in milk the greatest danger comes from localized tuberculous disease of the udder of the cow, or some other method of outside milk contamination, rather than through the excretion of tubercle bacilli in milk.

The *urine,* the *feces* and *purulent discharges* from tuberculous patients may be sources of infection, although this danger is believed to be slight because of the great dilution of the bacilli and because of the manner in which these discharges are ordinarily disposed of.

*Portals of Entry.*—Tuberculous infection enters the body through the nasopharynx, the bronchial mucous membranes and the digestive tract. This latter route is much more common in the child than in the adult. The passage of tubercle bacilli through mucous membranes may be effected without producing injury or disease of the parts through which they pass. The accidental contamination of vaccination, circumcision and other fresh wounds can only occur where gross carelessness leads to direct inoculation, and this is fortunately a very rare occurrence.

*Exposure.*—Tuberculosis is such a pandemic disease that practically every individual is exposed many times to its contagion. But this does not militate against the fact that the greatest danger comes from repeated exposure to the contagion in places especially infected with tubercle bacilli. In other words, one may say that, *other things being equal, the danger of contracting tuberculosis is in direct proportion to the frequency and size of the dose of the contagion.* This is a fact that should be impressed with especial force on the minds of the laity, since to them the contagion is

not apparent, inasmuch as active symptoms do not commonly develop, in infants and young children, until long enough after the exposure to the contagion for them to fail to see and recognize the connection between the exposure and the subsequent development of tuberculosis. If active tuberculosis followed as quickly upon exposure to the contagion as do diphtheria and scarlet fever, the laity would then quickly recognize the fact and insist upon such measures of isolation, quarantine and disinfection as would greatly reduce the prevalence of this disease.

Tuberculosis is very rarely contracted *in utero* by direct transmission through a tuberculous placenta; about forty such cases have been reported. It is not proven, however, that this disease can be transmitted by tuberculosis of the spermatozoa of the male or the ovum of the female without the intervention of a tuberculous placenta.

Heredity.—Heredity has from the earliest times been believed to be a most important factor in producing tuberculosis. At present, however, in the sense that one means that the patient has inherited a specific susceptibility to tuberculosis, heredity is believed to be a very unimportant predisposing factor, that is to say the special tuberculous diathesis which was supposed to furnish a favorable soil for the tubercle bacillus, while not wholly a negligible, is a comparatively unimportant factor in its spread. The hereditary factor is not so much a specific tendency to this disease as it is a weak constitution which belongs to the puny offsprings of weak and tuberculous parentage. Children of this class are usually anemic, malnourished and unable to offer the normal resistance to the tubercle bacillus. The question therefore of family tuberculosis is largely a matter of infection. Over 50 per cent. of all patients with this disease give a history of other cases in the family. All children inherit a defensive mechanism which enables them, under favorable conditions, to combat more or less successfully the contagion of tuberculosis. This defensive mechanism, which is very weak in infancy, becomes gradually stronger as the child grows older. It may be weakened by inheritance or by disease.

Acute Infectious Diseases.—Acute infectious diseases, especially those which involve mucous membranes and produce lymph-node enlargement, such as measles, whooping-cough, influenza and enteritis, are very important predisposing factors in preparing the soil for tuberculous infection, and are also important in developing a latent into an active tuberculosis.

Poverty.—Tuberculosis is one of the greatest causes of poverty. Comparatively prosperous families of the working class are, within a short period of time, reduced to abject poverty by reason of the fact that the productive member of the family is incapacitated from work by this disease. This leads to bad hygienic surroundings, insufficient food, overcrowding and the rapid dissemination of tuberculosis among the other, and especially the younger, members of the family. Here the poverty caused by tuberculosis becomes a most important factor in its dissemination. The winter season, by crowding poor families into dark and un-

wholesome surroundings, deprives them of sunlight and fresh air and promotes the spread of this disease by diminishing the normal resistance of the child and increasing its opportunities for contagion.

School Infection.—School infection is not as great a factor in spreading tuberculosis as it is in disseminating other contagious diseases. The protection here lies in the fact that a very great majority of the cases of tuberculosis in childhood are not characterized by active pulmonary symptoms, are not accompanied by the expectoration of tuberculous sputum, and are therefore not anything like so contagious as they are in adults, in whom the open pulmonary form is the common type of the disease.

Age.—Age is the most important of all the predisposing factors. The great majority of the cases of tuberculosis are contracted in childhood, even though the active symptoms may not occur until much later in life. The normal adult is practically immune from this infection; the vast majority of the cases of adult tuberculosis are the result of infection in childhood. Infancy is the most susceptible of all ages and perhaps there is no such thing as immunity during this period of life. If the young infant is repeatedly exposed to the contagion it will almost surely contract the disease, and yet the first few months of life show comparatively few cases. This may perhaps be due to the facts that during this time the opportunities for infection are less, and the breast milk upon which most infants are fed confers a partial immunity. As the child grows older its power of resisting the tuberculous infection becomes greater, and the disease when contracted usually runs a much less virulent course. The increasing resistance of the tissues of the child to tuberculous processes does not result in less frequent infection, but it does result in localizing and diminishing the severity of tuberculous processes. The mortality returns do not give a correct idea of the prevalence of tuberculosis at different ages in children. In infancy the death rate is high, and represents a large percentage of all the cases occurring at this age; latent and chronic forms of the disease rarely occur in infants. In childhood the death rate, compared with the number of cases, is small; at this age most of the cases are chronic, and the disease may last for years. During childhood the prevalence of tuberculosis gradually increases, and the severity of the disease gradually diminishes; the prevalence increases more rapidly and the severity diminishes more rapidly during the first three years of life than later. The frequency of tuberculosis in childhood has been a matter of much speculation since the mortality returns cannot be depended upon to determine the prevalence of this disease during this period of life.

It is, however, generally admitted that at least 20 per cent. of all children living in cities have tuberculosis in a more or less active form. In many of these, however, the disease is running a very insidious course in the deep-seated lymphatic glands. Tuberculin skin reactions indicate that a much larger percentage carry tuberculous foci in a latent form; these cases may present absolutely no clinical symptoms. Examinations made at dispensary clinics and institutions for the care of children show that, among

the children of the poor, from 60 to 80 or 90 per cent. react to the tuberculin skin tests; this determines the enormous prevalence of latent tuberculosis in childhood. [1] In 1895 the author wrote as follows: "Concealed lymph-node tuberculosis is the characteristic tuberculosis of childhood and is more prevalent than all the chronic diseases of childhood taken together. It is all about us every day; in our asylums, our schools, and our homes, masquerading as a preturberculous condition, as anemia, neurasthenia, lithemia, malaria and other ill-defined conditions; but in the meantime it progresses apace, and too often only casts off its disguise after irreparable damage has been done."

**Pathology.**—The tubercle bacilli find their way into the body through the portals of entry previously described, and in infancy and early childhood usually find lodgment in the bronchial, cervical or mesenteric lymphatic glands. Here they may be destroyed, or they may at once set up an active tuberculosis either at the point of entry or in some distant part of the body or, what is much more common, they may remain for months or years either dormant or in a slow state of incubation, until favorable conditions cause them to develop an active tuberculous inflammation.

The initial and characteristic lesion produced by the tubercle bacillus is the miliary tubercle, which is a minute grayish, translucent, firm nodule, about the size of a millet seed, which can be rather readily seen with the naked eye. They are made up of epithelioid cells often arranged in concentric layers, with occasional giant cells in their center; no vascular structures have been observed in them; tubercle bacilli are found in and among these cells. Under the influence of these bacilli, the tubercles multiply in number, become the center of active inflammatory changes, coalesce, and the oldest tubercles increase in size and commence to undergo a central necrosis which gradually transforms them into a cheesy mass. The spread of the tuberculous process may then occur by continuity and contiguity of tissues to neighboring parts, or by lymph and blood channels to more distant organs, thus producing the various forms of tuberculosis in infancy and childhood. A short sketch of the individual pathological lesions of these various types of tuberculosis here follows:

Lymph-Node Tuberculosis.—Lymph-node tuberculosis is by far the most common form in infancy and childhood. The initial lesion in the vast majority of instances not only begins in these glands but usually remains there for some time before the infection spreads to other tissues. The bronchial lymph nodes are of vastly greater importance as tuberculous foci because they are more frequently affected than any other lymph nodes in the body and because disease of these glands is of much more serious import in the spread of the disease to other and more vital tissues and because, by reason of their location in the bony cavity of the chest, their enlargement may produce severe pressure symptoms on the blood vessels, nerves and bronchi with which they are in close association. The bronchial lymph nodes, anatomically described as peritracheo-bronchial glands, are

[1] *New York Medical Journal*, August 10, 1895.

divided into three groups. The first group, or tracheal glands, are situated on both sides of the trachea, beginning on the right side in the angle of the trachea and right bronchus; they ascend along the trachea to the subclavian vessels and, beginning at a corresponding point on the left side of the trachea, they ascend to the arch of the aorta and the recurrent laryngeal nerves. These glands are in relation with the arch of the aorta, the recurrent laryngeal and pneumogastric nerves, the pulmonary artery and the superior vena cava. The second group is situated in the angle formed by the bifurcation of the trachea and extends along the large bronchi. They are in relation with the large bronchi, especially on the right, and with the esophagus, aorta and pneumogastric nerve. The third group extends along the bronchi into the lungs; they are associated with groups of glands in the angles of the bifurcation of the large bronchi as far as the fourth bifurcation, and with the veins and arteries which accompany the bronchi into the lungs. The anterior mediastinal lymph nodes are in relation with the right innominate artery, the right subclavian artery, and the arch of the aorta, and the posterior mediastinal lymph nodes are in relation with the esophagus and aorta. The cervical lymph nodes are very abundant and are in close association with the large vessels and nerves of the neck, but these nodes have little direct communication with the bronchial nodes, and their enlargement rarely produces pressure symptoms because they are not confined in a bony cavity. The superficial cervical lymph nodes below and behind the external ear and over the upper portion of the sternomastoid muscle, and the deeper cervical lymph nodes, in relation with the jugular veins, above and behind the clavicle, are commonly the site of tuberculous lesions. The mesenteric lymph nodes are very widely disseminated through the abdominal cavity and are in close association with the large nerves and blood vessels as well as with certain portions of the large intestine. A notable group is situated in the region of the appendix which is not uncommonly enlarged in mesenteric tuberculosis and forms a readily palpable tumor. The mesenteric lymph nodes, being unconfined by bony walls, do not usually by their enlargement produce pressure symptoms on the blood vessels and nerves with which they are in contact. But these nodes, unlike the cervical ones, are in close communication through lymph channels with the bronchial nodes, so that a tuberculous infection of these glands commonly leads to a secondary infection of the bronchial glands. On the other hand, bronchial lymph-node tuberculosis does not so commonly lead to infection of the mesenteric glands; this is perhaps explained by the direction of the lymph stream.

The agglutination and caseation of tubercles, with the resulting inflammatory changes, may cause great enlargement of lymph nodes and necrosis of lymphatic tissues. This destruction gradually involves neighboring nodes and the intima of adjacent lymph and blood vessels. In this manner masses varying in size from a pea to a hen's egg may be formed, producing mechanical as well as septic symptoms. An important fact in

the pathology of lymph-node tuberculosis is that, even after the deep-seated bronchial and mesenteric lymph nodes have undergone more or less caseation, nature, as a rule, succeeds in encapsulating, absorbing, and calcifying them, thus preventing further infection by the discharge of tuberculous pus through ulceration into the blood vessels, the bronchi, the trachea, the esophagus, the pleura, the pericardium, the peritoneum, or the surrounding cellular tissues. While all of these may occur they are comparatively uncommon, and recovery without ulceration after caseation of deep-seated lymph nodes is the rule rather than the exception; but caseation of ex-

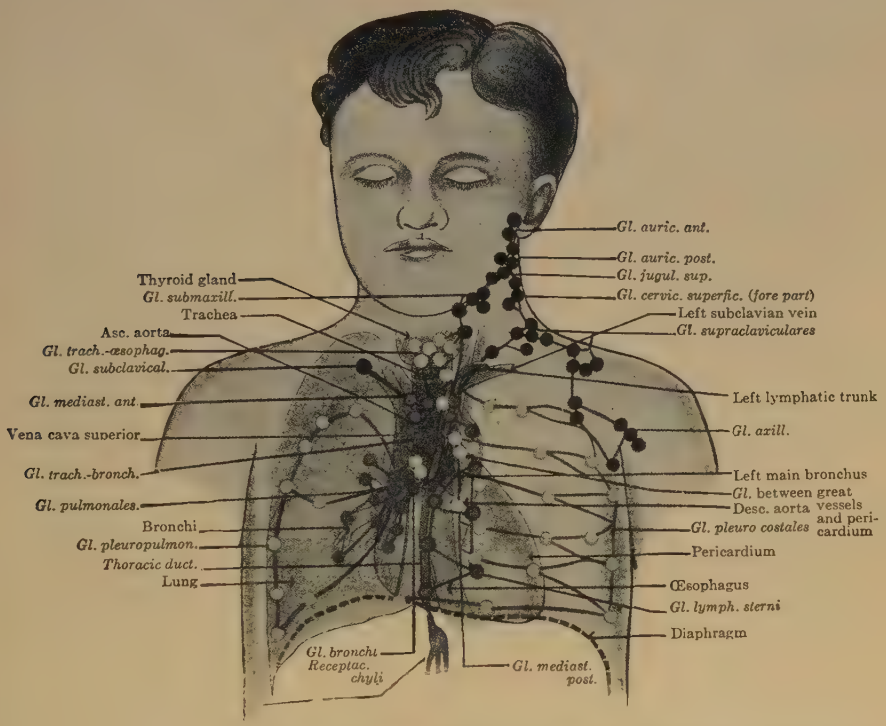



Fig. 61.—Bronchial and Other Lymph Nodes Mainly Affected in Tuberculosis. (Pfaundler and Schlossmann.)

ternal lymphatics in the neck, the groin, the axilla and elsewhere is more commonly followed by ulceration and the discharge of pus externally.

Pulmonary and Pleural Tuberculosis.—The lungs and pleura are not usually the site of the primary lesion in the tuberculosis of infants and young children; these organs are, however, as a rule, involved secondarily in all forms of severe tuberculosis. The lungs especially are almost always involved in well-advanced tuberculosis, and in the later stages of this disease are commonly the site of widespread and destructive lesions. These lesions follow mainly two general types, a widespread dissemination throughout the lungs and pleura of gray miliary tubercles accompanied

by an intense congestion and hyperemia of the involved parts; or localized patches of yellow, caseating tubercles which coalesce, break down, and discharge their purulent contents into the bronchi or, more rarely, into the pleura, thus forming small cavities scattered throughout the lungs. If the pulmonary process is a chronic one more or less fibrosis may occur around the diseased areas, resulting in encapsulation, absorption and cicatricial contraction. Tuberculosis of the pleura, which is usually associated with pulmonary tuberculosis, produces thickening of that membrane and fibrous adhesions which may interfere with pulmonary expansion. Empyema may occur. The most marked differences between the pulmonary lesions of infantile and adult tuberculosis are as follows: Infantile pulmonary tuberculosis is more disseminated; begins in the middle and lower lobes rather than in the apex; is commonly secondary to tuberculosis of

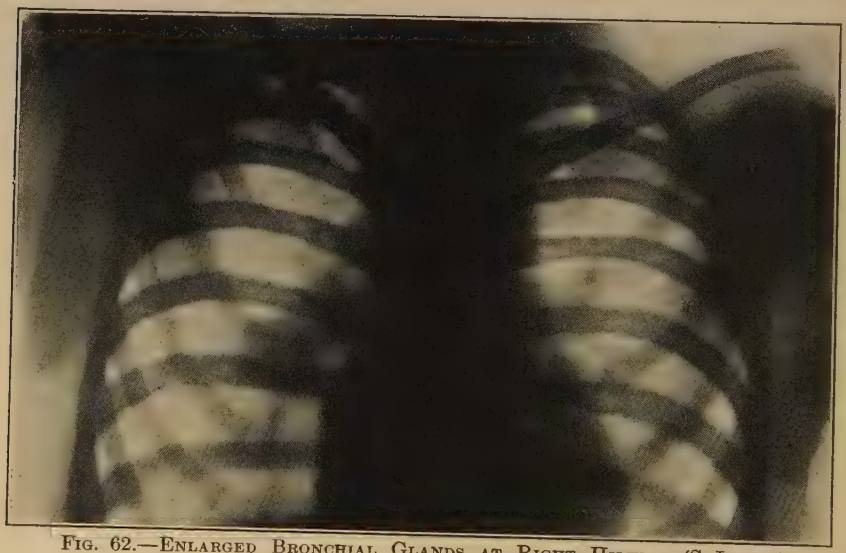


FIG. 62.—ENLARGED BRONCHIAL GLANDS AT RIGHT HILUM. (S. Lange.)

the bronchial lymph nodes, and is always accompanied by an active tuberculosis of these glands.

INTESTINAL TUBERCULOSIS.—Intestinal tuberculosis may be secondary to mesenteric lymph-node tuberculosis, or it may be primary, due to the swallowing of tubercle bacilli. Primary intestinal tuberculosis is a rare lesion, according to Bovaird, who, combining his own cases with those of Holt and Northrup, found only five primary intestinal cases out of a total of 369 cases upon whom careful autopsies had been made. Intestinal lesions usually begin with the formation of miliary tubercles in Peyer's patches; they multiply, coalesce, break down, and produce ulcers; these ulcers may gradually increase in size until they run together, forming long, ulcerated patches lying opposite to the mesenteric attachment; in healing they may cicatrize, causing more or less contraction of the intestine in its long axis.

Tuberculous Peritonitis.—Tuberculous peritonitis is secondary to intestinal or peritoneal lymph-node tuberculosis; the three conditions are very commonly associated. In peritonitis the peritoneal membrane is studded with tubercles, which set up a more or less active inflammation that may result in an exudation of fibrin, binding together the intestinal coils, the omentum and abdominal peritoneum, so that the whole peritoneal cavity may be obliterated; in other instances the exudation may be serofibrinous, resulting in a certain amount of agglutination of the intestinal coils, with a greater or less quantity of serum in the peritoneal cavity, thus producing ascites; or the tubercles may adhere, forming large masses which disintegrate, producing pockets of pus; these may ulcerate and discharge their contents through the abdominal wall or into the intestine. The writer once observed a case of this kind which ulcerated through the intestine and also through the umbilicus, forming a fecal fistula, which persisted for a number of months until the child's death.

General Miliary Tuberculosis.—General miliary tuberculosis is caused by the discharge of tuberculous pus into the blood or lymph streams. The tubercle bacilli are thus widely distributed throughout the body and find a lodgment in various organs, especially the lungs, liver, spleen, kidneys and brain. Miliary tubercles are soon widely disseminated through these organs and later every organ in the body may be involved. Around these tubercles is a small congested inflammatory area, especially marked in the lungs. The liver and spleen are enlarged.

Bone and Joint Tuberculosis.—Bone and joint tuberculosis is secondary to tuberculous foci elsewhere, and the contagion is carried by the blood and lymph channels to these structures. This manifestation of tuberculosis in the vast majority of cases begins in childhood, and is one of the common manifestations of this disease. The bones most frequently affected are the vertebræ, the short bones of the hands and feet and the epiphyseal portions of the long bones. Rotch's table of 3,820 cases shows the relative frequency with which the various joints of the body are attacked.

*Rotch's Table from the Children's Hospital, Boston.*

| | |
|---|---|
| Spine | 1964 |
| Hip | 1402 |
| Ankle | 300 |
| Knee | 104 |
| Wrist | 20 |
| Shoulder | 15 |
| Elbow | 15 |

This form of the disease begins by a deposit of tubercles in the cancellous tissue of the bone near the joint, and there result a grayish-red infiltration and inflammation which may be followed by caseation with the destruction of bony tissue and perforation into the joint, producing a purulent synovitis. The joint surfaces are usually involved before caseation of the bone takes place, and there are produced a sero-fibrinous exuda-

tion into the joint cavity and a deposit of tuberculous granulation tissue on the synovial membrane; this granulation tissue may caseate and thus form a purulent synovitis. It is important, however, to remember that both bone and joint tuberculosis in their earlier stages commonly yield to rational medical and surgical treatment.

**Symptomatology.**—In studying the symptomatology of tuberculosis the following clinical types of this disease will be considered in the order named:

Tuberculosis of lymph nodes.
General miliary tuberculosis in infants.
General miliary tuberculosis in older children.
Tuberculous bronchopneumonia in young children.
Tuberculosis of lungs in older children.
Tuberculous peritonitis.
Tuberculosis of bones and joints.
(Tuberculous meningitis is studied with the other forms of meningitis.)

Lymph-Node Tuberculosis.—[1]This form of tuberculosis, when it involves other than the cervical lymph nodes, is so obscure and so commonly overlooked that it is of the utmost importance that the physician should always keep in mind its prevalence and be constantly on the lookout for its signs and symptoms. Many years ago I expressed the opinion, now generally concurred in, that pronounced *simple anemia,* occurring in young children with a history of exposure to tuberculous contagion, was strongly suggestive of concealed lymph-node tuberculosis, and upon these conditions alone one was warranted in making a tentative diagnosis of tuberculosis and putting the child upon the treatment for this disease. The type of anemia which occurs in tuberculosis is a simple secondary anemia of the chlorotic type.

*Neurotic disease* in children is very frequently an indication of concealed lymph-node tuberculosis. Over 34 per cent. of the last 400 tuberculous patients taken from my dispensary records show well-marked neurotic disease, such as chorea, incontinence of urine, hysteria, general nervous irritability and night terrors. A well-marked neurosis therefore occurring in a child without apparent cause should lead the physician to search for other symptoms of tuberculosis. Tuberculous children of this type are commonly precocious. The precocity, however, which is associated with concealed tuberculosis, is fitful and cannot be long sustained. In the beginning of the school year these children may make a brilliant showing, but they usually break down in the latter half of the year with well-marked neurotic disease associated with anemia and general physical weakness. In my dispensary records 45 per cent. of all neurotic children are actively tuberculous.

*Dyspnea* and *pain* in the side are in my experience frequently associated with bronchial lymph-node tuberculosis, even before there is any clear evidence of pulmonary tuberculosis. These symptoms are aggravated

[1] *New York Medical Journal,* August 10, 1895.

by exercise, and, like the anemia and nervous symptoms, are of much greater significance when they occur in children with a tuberculous family history.

Respiratory Symptoms.—Tuberculous children catch cold readily and often suffer from frequent attacks of snuffles and nasal catarrh. The nasal discharge may be irritating and produce a slight eczema and thickening of the lip, giving a more or less characteristic expression. They may also suffer from frequent attacks of bronchitis even before an active pulmonary tuberculosis can be demonstrated.

*Abnormal dwarfishness* may be an evidence of concealed tuberculosis. By an abnormal dwarf is meant not only one that is underweight, but one that also lacks symmetry in development. The relation of weight and girth of chest is of special importance in this particular. A marked disproportion between the weight and height, when associated with poor chest development and a family history of tuberculosis, should prompt a careful search for other signs of bronchial lymph-node tuberculosis.

*Progressive failure of health, loss of weight, or even failure to gain in weight,* which in the growing child is equivalent to loss of weight in the adult, is frequently a symptom of lymph-node tuberculosis, and when these conditions exist without apparent cause tuberculosis should be suspected.

The early appearance of and irregularity in the *menstrual function* commonly occurs in young girls suffering from lymph-node tuberculosis. Of 52 girls who were irregular in their menstrual function, 47 gave family histories of tuberculosis and 5 gave family histories free from tuberculosis. Of 110 girls who were regular in their menstrual function, 80 gave non-tuberculous family histories, and 30 gave family histories of tuberculosis. A family history of tuberculosis implies also the more important fact that there was exposure to the tuberculous contagion.

If the mesenteric lymph nodes be tuberculous, and they are rarely involved independently of the bronchial lymph nodes, we may have, to a greater or less degree, associated with the above symptoms dyspepsia with a tendency to *chronic diarrhea.* Obstinate diarrhea and other gastrointestinal disturbances may occur in this variety of tuberculosis even before lesions appear in the intestinal mucosa; enlargement of the spleen is commonly associated with these symptoms.

*Fever* may or may not be present in lymph-node tuberculosis, but where the disease is at all active a slight rise of the temperature may usually be discovered, and in aggravated cases it may rise regularly to 103° or 104° F. some time during the day. The temperature in these cases produces little or no discomfort, so that it is not unusual for a child with a temperature of 103° F. to protest that there is nothing the matter with it. Night sweats may be present, even though there be but a slight rise of temperature. They are associated with the anemia, nervousness and malnutrition of lymph-node tuberculosis.

Friedlander has called attention to the fact that a relative and ab-

solute *lymphocytosis* occurs in lymph-node tuberculosis, and when this fact is taken in connection with the well-established fact that certain other diseases, such as whooping-cough, also produce lymph-node enlargement, and are accompanied by a lymphocytosis, he suggests that this sign may be a valuable one in testifying to the existence of an inflammation of the lymph glands in cases where concealed tuberculosis is suspected.

*Enlargement of external lymph nodes* in the groin, axilla, neck and elsewhere may be a very important sign of the existence of bronchial or mesenteric lymph-node tuberculosis. The presence of enlarged external lymph nodes, which can be easily seen and felt, if associated with the symptom group previously described, is of the greatest value in confirming the diagnosis of concealed tuberculosis, but it must be remembered that a very advanced stage of bronchial lymph-node tuberculosis may exist with little or no enlargement of external lymphatics, and it must also be remembered that a very extensive lymph-node tuberculosis may occur in the cervical and other superficial lymphatics with little or no involvement of the deep-seated bronchial or mesenteric lymph nodes. The extent of the disease, therefore, in external lymphatics bears absolutely no relationship to the extent of the disease in deep-seated lymphatics.

*Skin tuberculides,* described by Hamburger, may appear as small, red or brownish papules, perhaps three or four in number, scattered over the body. They soon become covered with a small crust, which on removal leaves a depression. In this crust tubercle bacilli may be found.

*Physical signs* are rather unreliable because of their variability. Great enlargement of the bronchial lymph nodes may exist without producing physical signs which will lead to their detection, but in some cases the physical signs are of importance. Percussion may elicit dullness over and on either side of the manubrium sterni, and on either side of the spine in the interscapular region; the dullness is more frequently found on the right side. Auscultation is even less reliable than percussion. Grancher believes that the harsh breathing sounds, which are normal in the right apex of the child, when greatly exaggerated, are a sign of importance. The bronchovesicular breathing, with prolonged and harsh inspiration, which may be heard in some instances at the apex, is especially important if it occurs on the left side. Feeble breath sounds over the whole of one lobe are a sign of significance. A venous hum, as noted by Eustace Smith, may sometimes be heard over the manubrium if the head of the child is bent backward so that the enlarged gland will compress the left innominate vein against the sternum.

Palpation in mesenteric lymph-node tuberculosis is of great value, since an enlarged spleen may usually be found and deep-seated lymph glands, especially in the region of the appendix, may be made out. In bronchial lymph-node tuberculosis, however, palpation is of little value, since these glands cannot be felt. Deep palpation, however, in the episternal notch beneath the clavicle, may reveal enlarged glands, which may be associated with the bronchial chain.

PLATE IV.

THE VON PIRQUET TUBERCULIN SKIN REACTIONS.
(From Hamill, Carpenter and Cope).

*Pressure signs* produced by enlarged lymph nodes are at times a signal aid in confirming the diagnosis of bronchial lymph-node tuberculosis. Hall, in a comprehensive review of the literature of this subject, has called special attention to the value of these signs. A severe paroxysmal cough, resembling pertussis, occurring more frequently at night, and often associated with asthmatic breathing, is a common and very significant symptom. Pressure on the trachea may produce tracheitis, inspiratory dyspnea and cyanosis. Pressure on one bronchus, most commonly the right, may produce bronchitis and a diminished expansion, and feeble vesicular breathing over that portion of the lung to which the bronchus leads. Pressure on the esophagus may produce difficulty in deglutition. Compression of blood vessels produces venous-stasis with edema of the face and arms, and pressure on the pulmonary veins may produce congestion of the lungs. Pressure on the recurrent laryngeal nerve may produce a hoarse, harsh cough and even aphonia.

*Radiographic examination* of the chest may reveal the existence of enlarged bronchial lymph nodes and may be of value in confirming the diagnosis. If the lung be involved patches of consolidation and limitation of motion at base of diseased lung may be seen.

*Tuberculin reactions* in recent years have come to be considered of great value in confirming the diagnosis of concealed tuberculosis. They are so sensitive that they reveal minute foci of tuberculosis in the latent or inactive forms of this condition. The presence of these reactions therefore does not always mean an active tuberculosis. The severity, however, of the reaction and the rapidity with which it occurs may be of some value in determining the degree of activity of the tuberculous process. This rule also has its limitations, since these reactions fail to appear in advanced cases, and their value lies not in making a differential diagnosis in the acute and grave forms of tuberculosis, but in determining the presence of tuberculous foci in suspected cases of chronic concealed tuberculosis. In these the reaction rarely fails, and a negative result would mean the absence of tuberculosis. Of these tuberculin reactions, the Moro ointment test is the simplest of application and is sensitive enough for all practical purposes. The von Pirquet scarification test is also simple in its technique and more sensitive in its reaction. The conjunctival test is now rarely used, because it involves slight danger to the eye. The subcutaneous test presents no advantages over the others, is more complicated, and offers greater opportunities for infection. The technique of these tests is elsewhere given.

**Diagnosis.**—From the foregoing outline it is evident that the diagnosis of concealed lymph-node tuberculosis is not only possible, but that the failure to make this diagnosis rather early in the disease indicates a lack of diagnostic skill and knowledge on the part of the attending physician. If the fact is kept in mind that it is the most common of all the chronic diseases of childhood, and that a history of possible exposure to the tuberculous contagion may have occurred months and even years before the

active symptoms are developed, the physician will then be prepared to interpret the syndrome above outlined. Anemia, neurotic disease, general malnutrition, dyspnea and pain in the side on exercising, proneness to catch cold, frequent attacks of bronchitis, abnormal dwarfishness, progressive failure of health, loss of weight, chronic diarrhea with enlargement of the spleen, slight intermittent fever, lymphocytosis, enlargement of external lymph nodes, or paroxysmal cough resembling pertussis, occurring in a child between the ages of two and fifteen, should lead to a tentative diagnosis of concealed lymph-node tuberculosis, which may be confirmed or disproven by a careful physical examination, the subsequent history of the case, and, if necessary, by tuberculin skin reactions and radiographs.

Tuberculous Cervical Adenitis (Scrofula).—Tuberculous cervical adenitis is almost as common as tuberculous bronchial adenitis; the two conditions, however, present altogether distinct symptom groups. The cervical lymph nodes are not in close anatomical connection with the bronchial glands. The clinical facts that bronchial adenitis is very common and cervical adenitis very rare in infancy, and that cervical adenitis in the older child often occurs without evidence of bronchial adenitis, confirm the belief that there is little direct communication between these two groups of glands. The clinical picture of cervical adenitis may be quite independent of bronchial adenitis, and the existence of the two symptom groups in the same child means, in the majority of instances, that the child has an independent infection of the two groups of glands rather than that the infection has traveled from one group to the other. Tuberculous cervical adenitis is essentially a disease of childhood; it is comparatively rare in the infant and adult. The great majority of the cases occur between the third and fifteenth year of life.

The diagnosis of cervical adenitis is a very simple matter. The lymph nodes involved are readily palpable and the only question which may arise is as to whether their enlargement is due to an inflammation produced by tubercle bacilli or other microorganisms. In tuberculous cervical adenitis the process is essentially a chronic one and the glands are less tender. When suppuration occurs through the skin the reparative processes are slower and there is more tendency to sinus formation and to scar tissue. There are also very frequently blepharitis, phlyctenular keratitis, coryza, chronic nasal catarrh and eczema of the lip and face. A positive diagnosis of the character of the microorganism producing the adenitis cannot always be made without dissecting out the gland (which is the best treatment in troublesome cases), and subjecting it to a microscopical examination or injecting it into a guinea-pig. Because of the fact that the great majority of the chronic cases are tuberculous and because of the importance of instituting proper treatment in these cases, it is wise for the physician to treat all such cases as tuberculous. When the disease is confined to the cervical glands the constitutional symptoms are not marked; if pronounced anemia and severe malnutrition are present the inference

is that the bronchial or other deep-seated lymph nodes are involved. Tuberculous cervical adenitis usually manifests itself by enlarged glands in the sub-maxillary region, varying in size from a hazelnut to a walnut; these may coalesce and form large, solid tumor masses. Suppuration may occur, with the discharge through the skin of curdy, cheesy pus, forming a sinus which remains open or is only temporarily closed, until the whole of the glandular tissue involved is disintegrated and discharged. Following and accompanying this process the skin may be marked by large, rough, unsightly scars. Cervical adenitis may be aggravated, or even caused by disease of the adenoids, tonsils and pharynx.

General Miliary Tuberculosis in Infants.—This is a very insidious disease, presenting, as a rule, only the symptoms of general marasmus. The infant commences to lose in weight and strength, and is anemic. A slow wasting and failure in health, without apparent cause, is the dominant symptom. This condition is commonly mistaken for some disease on the part of the gastrointestinal canal. The infant, as a rule, takes but little food and has secondary digestive disturbances, such as regurgitation of food, vomiting and diarrhea, and the stools may show lack of digestion and assimilation. After a longer or shorter time, usually some months, there is fever, which may be constant or intermittent, the spleen and liver are enlarged, the digestive disturbances are increased and the lungs commence to show evidence of bronchitis and then bronchopneumonia. The terminal symptoms are continuous fever, more or less cough, rapid pulse and great prostration. Death may result from general exhaustion, resembling marasmus, from a terminal bronchopneumonia or tuberculous meningitis.

General Miliary Tuberculosis in Children Over Five Years of Age.—This disease is always secondary to tuberculous foci elsewhere in the body, which have ulcerated into the blood or lymph streams and produced the general infection. These previous foci may be located in the lungs or the bones, but in the vast majority of instances they are in the lymph nodes, and this is the reason why this form of tuberculosis is nearly always preceded by the symptoms of lymph-node tuberculosis. This fact cannot be too strongly insisted upon. Following the symptoms of lymph-node tuberculosis previously given, the child becomes acutely ill with a continuous fever, marked by general prostration and progressive emaciation, causing a clinical picture closely resembling that of typhoid fever in the adult. The two conditions, however, should not be confused. In general miliary tuberculosis the fever, although continuous, is not as regular as that of typhoid, the spleen is not so frequently enlarged, digestive disturbances are not, as a rule, marked, rose spots are absent, the Widal reaction is negative, and as the disease progresses the fever does not abate at the end of the third or fourth week, but continues with increasing prostration, emaciation, and cachexia. A tuberculous bronchopneumonia or meningitis may terminate the clinical picture.

Tuberculous Bronchopneumonia.—This condition occurs most com-

monly in young children between the ages of two and five. It may be the terminal picture of the marantic type of general miliary tuberculosis in the infant. In the child it is much more commonly the sequel of a bronchial lymph-node tuberculosis which has existed for months or even years and has been finally developed into a tuberculous bronchopneumonia by an attack of measles, whooping-cough, influenza, or bronchitis. The temperature chart (page 460) indicates the course the fever may take and also the increase in respiration as the disease progresses. Cough, dyspnea, cyanosis and great prostration are present. Tubercle bacilli can usually be demonstrated in the sputum, which is caught by wiping out the pharynx with a piece of gauze during an attack of coughing. The disease in younger children may run from two to four weeks and is almost invariably fatal. In children from four to six years of age it may last six or eight weeks, with great variations in the severity of the symptom group. The apparent improvement which so often occurs in these cases is very misleading, since an acute exacerbation of the symptoms, as a rule, quickly follows, and the disease progresses to a fatal termination. In some instances, however, especially in older children, one of these intervals of apparent convalescence may be prolonged into an actual convalescence, and the child is again slowly restored to health.

The physical signs are those of acute bronchopneumonia elsewhere described. Râles of various kinds may be heard over the lungs. Over small areas crepitant râles, diminished resonance and bronchovesicular breathing may be found. The physical signs, however, may occur late and are often very elusive. The diagnosis is therefore commonly made by the tuberculous family history; previous or present symptoms of tuberculosis elsewhere in the body; the onset of the disease after one of the acute infections; the presence of tubercle bacilli in the sputum, and the symptoms and signs of an acute bronchopneumonia, running, especially in slightly older children, an irregular and prolonged course.

Tuberculosis of Lungs in Older Children.—Pulmonary tuberculosis, in children between the ages of six and fifteen, is usually preceded by and almost always associated with bronchial lymph-node tuberculosis. The symptomatology, therefore, of the two conditions is inseparably associated. In the great majority of instances there is a longer or shorter interval of time in which the symptoms of bronchial lymph-node tuberculosis are present before the pulmonary symptoms can be noted. In other instances the infection of the lung and the lymph nodes may be almost coincident, and in these cases the two symptom groups may be combined from the beginning of the disease. The pulmonary disease, extending, as it commonly does, from the bronchial lymph nodes, involves the middle portions of the lungs, extending first to the upper lobes and after a time involving the lower lobes.

The symptoms are those of bronchial lymph-node tuberculosis, associated with recurring attacks of tuberculous bronchitis, or bronchopneumonia. After a time the tuberculous process localizes itself more definitely

in the lungs, and then takes the form and presents the symptoms and physical signs of phthisis in the adult. The resistance to the progress of this disease is not, however, as great in the child as it is in the adult, and it therefore runs a more rapid course and has a more unfavorable prognosis.

The diagnosis in these cases is made by the family history, the preceding or accompanying symptoms of bronchial lymph-node tuberculosis,

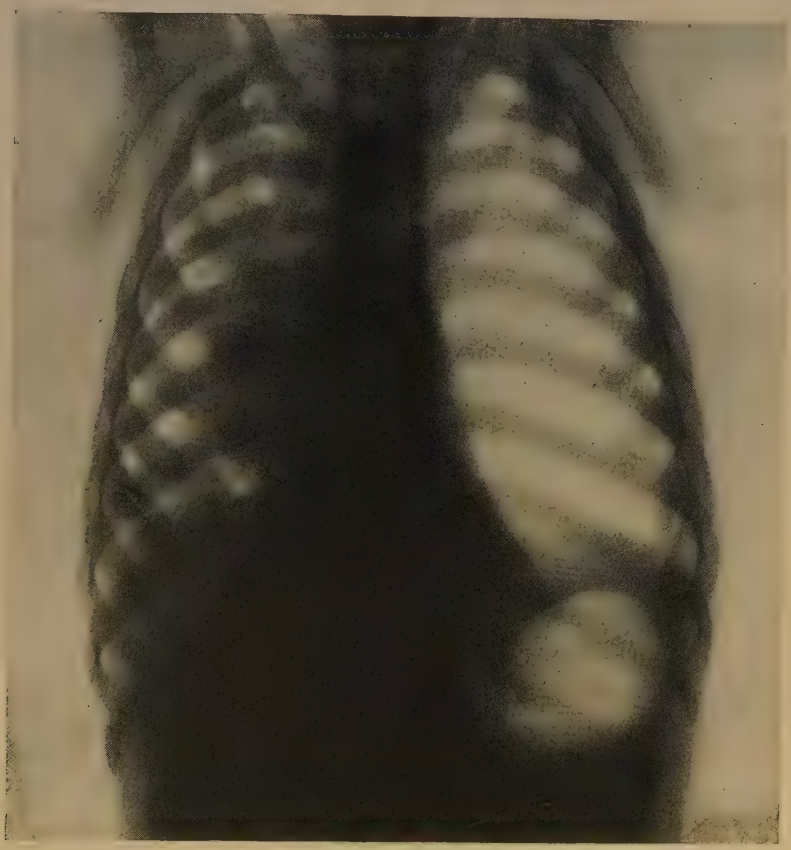


FIG. 63.—PULMONARY TUBERCULOSIS WITH LEFT-SIDED PNEUMOTHORAX. (S. Lange.)

the recurring attacks without apparent cause of bronchitis and bronchopneumonia, and later the physical signs of phthisis, and, most important of all, the finding of tubercle bacilli in the sputum. The sputum may be obtained by irritating the epiglottis, thus producing a cough, and as the mucus is brought up it is caught in the back part of the throat on a piece of gauze. This method of obtaining mucus is, as Holt has demonstrated, very successful; tubercle bacilli may be found in the mucus thus obtained in at least 70 or 80 per cent. of the cases of pulmonary tuberculosis in children.

Tuberculous Peritonitis.—Tuberculous peritonitis occurs most commonly between the fifth and the tenth year of life, and is usually secondary to mesenteric lymph-node tuberculosis; but it may also occur in the later stages of other forms of tuberculosis, especially the general miliary tuberculosis of infancy. The onset of this condition is commonly preceded, for a considerable period of time, by the symptoms of lymph-node tuberculosis. The characteristic symptoms of peritonitis are then slowly developed. There is abdominal tenderness with attacks of pain, vomiting, and constipation or diarrhea. The liver and spleen are usually enlarged. Abdominal distention, with resistance and induration, are

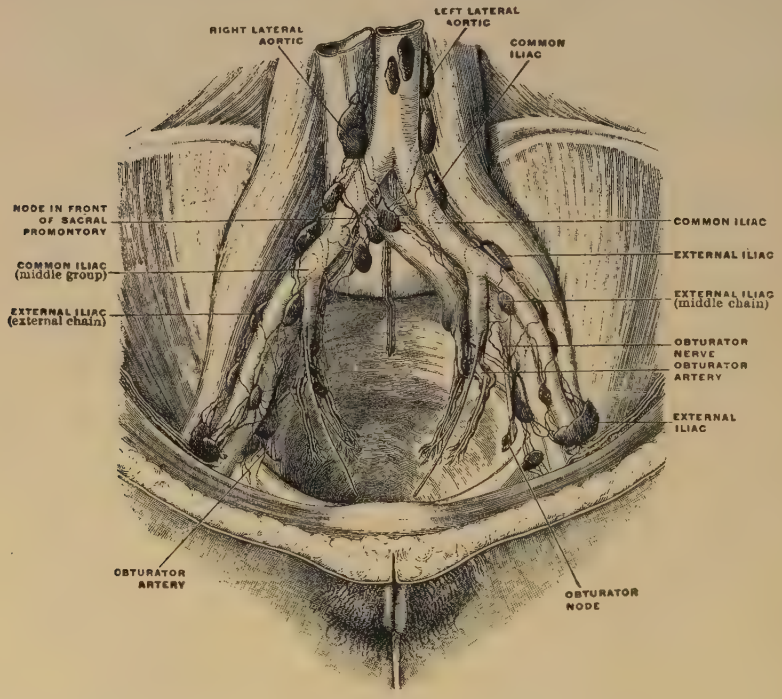



Fig. 64.—Ilio-pelvic Lymph Nodes. (Poirier and Charpy.)

marked, and these signs may be more or less localized, especially in the early stages. Increase in the abdominal distention, with or without ascites, may occur. Tumor masses may be felt, especially in the right iliac region, about the head of the colon, and in the hypogastric region along the thickened omentum. Localized indurations may sometimes be demonstrated by rectal examination. As the process extends the abdominal induration becomes general, the abdominal distention more marked, and the emaciation of the arms, legs, face, and body more extreme. This phase of the disease may last for months or years with marked remissions and exacerbations in the symptom group, until death or a slow convalescence terminates the clinical picture. The fever of tuberculous peritonitis is

irregular in type and the fluctuations in the temperature curve vary with the activity of the tuberculous process. For all practical purposes it is safe to assume that every *chronic* exudative peritonitis is tuberculous, notwithstanding the fact that Henoch and other German writers have described a very rare form of simple or *non-tuberculous,* chronic, exudative peritonitis occurring in older children. From a clinical standpoint it matters little whether such a disease exists or not. If it does, it is admittedly a very rare affection and its treatment is the same as that of tuberculous peritonitis.

While the above clinical picture is the most characteristic and the most common one presented by tuberculous peritonitis, sharp variations may occur, especially in acute cases. The sudden onset, presenting the symptoms of fever, vomiting, constipation, abdominal distention and tenderness in the right iliac region, may closely resemble acute perforative appendicitis, and the differential diagnosis may depend upon the previous history or the findings of an exploratory incision. Less acute cases with fever, diarrhea, large spleen, abdominal distention, and right iliac tenderness may present a picture somewhat like typhoid fever. In these cases the differential diagnosis may depend upon the absence of rose spots, a negative Widal reaction, and the subsequent course of the disease.

Tuberculosis of Bones and Joints.—This is a common manifestation of tuberculosis in children between the ages of three and fifteen. It presents itself in a number of distinct clinical types, of which the most important are: Pott's disease, hip-joint and knee-joint disease and tuberculous dactylitis. All of these are surgical affections, but because of the importance of an early diagnosis their characteristic local manifestations are here briefly noted.

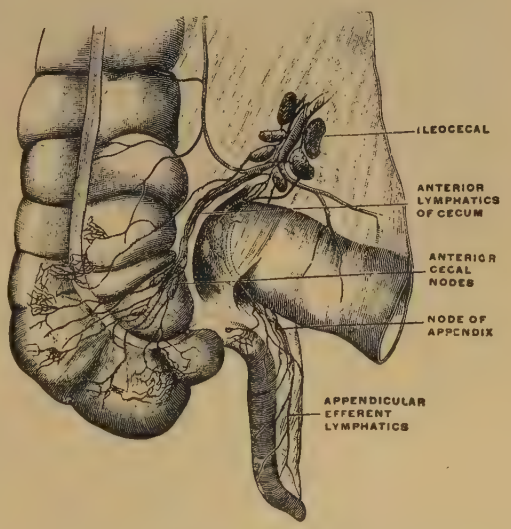



Fig. 65.—Lymphatics of the Cæcum and Appendix. (Poirier and Charpy.)

*Pott's disease* (caries of the spine) is a chronic tuberculous disease of the spine, characterized by the symptoms of lymph-node tuberculosis previously given, and by a localized stiffness, rigidity, pain, and tenderness in some portion of the spinal column. The pain is commonly referred to the parts supplied by the spinal nerves irritated by the diseased bone. During this stage the child assumes a position in walking and stooping which will relieve pressure on the vertebræ and keep the spinal column rigid. Later the spinal curvature, which

determines the diagnosis, makes its appearance. This is usually a sharp posterior curvature producing a characteristic deformity. It may be differentiated from rachitic and other spinal curvatures by its immobility, as demonstrated in the chapter on Examination of the Sick Child. Compression myelitis may result when the disease is in the upper half of the spine. If the cervical vertebræ be affected, the neck is held stiff, motion of the head produces pain, usually of a neuralgic type, involving the occipital region and sides of the neck. If the dorsal vertebræ be involved, the body of the child is held rigid and intercostal neuralgia and abdominal pains are complained of. If the lumbar spine is diseased the neuralgic pain extends into the legs and may be located in the hip or knee.

*Hip-joint disease* is a chronic tuberculous disease of this joint which, as a rule, is very insidious in its onset. It is, however, usually preceded and accompanied by the symptoms of lymph-node tuberculosis previously described. Lameness, sharp paroxysmal pain often referred to the knee, tenderness of the joint and disinclination to walk mark the onset. Tenderness may be elicited by pressing the joint surfaces together, and as this increases the lameness becomes more marked. Reflex muscular rigidity produces a stiffness of the joint or a limitation of its motions. The muscles of the thigh and calf show marked atrophy, and if the child is placed in an upright position the flattening in the gluteal fold of the affected hip and lack of symmetry of the two sides are very characteristic. As the disease progresses to the second stage the hip joint becomes fixed, the thigh flexed and somewhat adducted, due to contraction of the ilio-psoas muscle from muscular spasm. The cordlike contracture of this muscle is an important diagnostic sign. Lordosis and tilting of the pelvis occur. In the third stage the thigh is rotated inward, adducted, and the deformity of the hip becomes much greater. The leg is drawn up by muscular action, is several inches shorter than normal, and, while the hip may be much swollen, the lower portion of the thigh and leg are wasted. An abscess may form and point in Scarpa's triangle in the gluteal region or above Poupart's ligament.

Fig. 66.—Tuberculosis of Spine. (Pott's disease.)

*Knee-joint tuberculosis* is characterized by pain and a chronic "white swelling" of the joint. The joint motions are limited and the swelling is commonly boggy or gelatinous in character.

*Tuberculous dactylitis* occurs in the phalanges of the hands and feet. The swelling extends from joint to joint, is essentially chronic, is pyriform in shape and boggy in character.

An X-ray picture may be of great value in confirming the diagnosis of bone and joint tuberculosis.

**Prophylaxis.**—In April, 1896, I was asked to take charge of a tuberculous patient for the purpose of protecting her unborn child from tuberculosis. On the mother's side there was a family history of tuberculosis. The father was sturdy and his family history had no tuberculous taint. The mother was confined to her bed with an advanced stage of pulmonary and laryngeal tuberculosis.

The sick room, divested of all unnecessary hangings and carpets, was thoroughly cleansed; this was repeated at short intervals during the mother's long illness. A trained nurse was installed and instructed in the methods of destroying tubercle bacilli. The patient coughed into gauze napkins and the sputum was at once destroyed by fire. It was with the greatest difficulty that the mother was kept alive by hypodermic medication and such food and alcohol as she was able to take, until the baby was born in September, five months later. Previous to the birth of the child, a large and well-aired room on the same floor of the house as the mother's bedroom was selected. Everything was removed from this room, the paper was taken from the wall, the room repapered, the woodwork and floors scrubbed and washed down with a solution of bichlorid of mercury, and new furnishings supplied. On the birth of the child he was removed immediately to this room and placed in charge of a wet nurse. The mother lived nine weeks after the birth of her child and during this time, in deference to her self-sacrificing spirit, the infant was carried into the sick room once a day, remaining there for a few minutes only. But the mother, a very intelligent woman, did not nurse or fondle her child during this time. Before each daily visit of the infant to its mother's room, the windows of the sick room were opened and the room aired. On the death of the mother the whole house was carefully fumigated and cleansed. Every room was repapered, and the floors and woodwork scrubbed and washed down with bichlorid of mercury. The furniture was cleansed, the carpets and hangings all over the house were replaced by new ones and the bedding of the sick room was destroyed by fire. The infant, which at birth was very frail and malnourished, commenced to thrive and gradually grew stronger, so that when he was a few weeks of age he commenced his daily outings. From that time he was kept in the open air as much as possible, slept in a well-ventilated room and, when nine months of age, was gradually weaned and placed upon cow's milk. During the next five years of the child's life the fresh-air treatment was continued, and he was carefully fed within the range of

his digestive capacity; milk, eggs, meat, and cereals were utilized as much as possible in building up his nutrition; under this régime he continued to thrive in a normal manner. During this period he was carefully protected from all contagious diseases, especially tuberculosis, measles, influenza and pertussis. Between seven and nine years of age, however, he went safely through measles and pertussis, which he contracted at school. The boy is now seventeen years of age, is well developed physically, and is spending his winters in an Eastern boarding school and his summers in the open, camping out. At the time of the birth of this boy he had one sister, three and a half years of age, who lived in the same house and who was as carefully protected from tuberculosis during this time as was her younger brother. She is to-day a normal, sturdy girl of twenty.

This narrative portrays the underlying principles which should be adopted in the prophylactic treatment of tuberculosis, and illustrates what can be accomplished by the careful carrying out of these principles, without removing the child from its immediate tuberculous surroundings. It also most forcibly illustrates the axiomatic fact that the prevention of contagion in family tuberculosis is largely a question of money. The father in this instance had ample means, was a man of intelligence, and urged that no expense be spared in the protection of his children, and the result was a satisfactory one.

The prevention of tuberculosis, however, among the poor is altogether a different problem, and one that cannot be, or at least has not as yet been, satisfactorily solved. In dealing with an individual case the physician must therefore, within his limitations, carry out the following principles. Tubercle bacilli, from whatever source they may possibly come, must be destroyed by germicides or fire so as to prevent the contamination of the immediate surroundings of the patient. The sputum must be carefully collected and destroyed, and the room in which the tuberculous patient lives must be cleansed and disinfected as often as possible. Children should not be allowed to come in close contact with tuberculous individuals, as there is not only danger from the dried tubercle bacilli scattered about the room, but also from the tuberculous spray which is projected, by coughing, several feet into the surrounding atmosphere. This rule applies not only to the social and home life of the child, but also to its school life. Systematic school inspection is of importance in preventing the spread of tuberculosis and other infectious diseases. Kissing and fondling of children by tuberculous patients should be absolutely prohibited. Cow's milk should be obtained from a non-tuberculous herd of cattle, and its subsequent contamination by tubercle bacilli most carefully avoided, and where these conditions cannot be satisfactorily carried out the milk should be pasteurized for forty minutes at a temperature of 140° F.

An infant should under no conditions be allowed to nurse a tuberculous mother or wet-nurse, because such milk is not only likely to be innutritious, but because the child comes in such close contact with the

tuberculous nurse that there is great danger of its contracting the disease. Under ordinary conditions it is far safer to separate the young child from its tuberculous surroundings for at least a portion of the time, in this way diminishing the danger of contagion and placing the child in surroundings where it can get purer air and more sunshine, looking to its physical upbuilding and increased resistance to the tuberculous contagion; where this cannot be done it is advisable to remove the infected member of the family and clean up the surroundings.

Children with a tuberculous family history, which, as a rule, implies that they have had opportunities to contract the contagion, should be carefully guarded throughout their whole childhood from tuberculosis, measles, pertussis and influenza, the last three named diseases being especially potent factors in preparing the soil and opening the gateways for tuberculous contagion, and also in developing a latent into an active tuberculosis. Children of this class should, if possible, live in the country and spend their winters in some warm, dry climate, which will enable them to live an out-of-door life, but wherever they are located, in the city or in the country, under suitable or unsuitable climatic conditions, they should live in the open air and sleep in well-ventilated rooms, or *out of doors,* when possible. During all of this time they should have great care given them in the selection of their food. Eating good, nutritious food at proper intervals and indulging in outdoor sports will do much to develop a physique which gives the child an increased resistance to the tuberculous contagion. Because of the prevalence of tuberculosis all children, whether coming from tuberculous stock or not, should, to protect them not only from this, but from other contagious diseases, have careful attention devoted to diseases of the nose, pharynx, tonsils and adenoids. The eradication of diseased tissues in these locations will at least partially close the most common gateway, not only to tuberculosis, but to a number of other contagious diseases.

**Treatment.**—General Treatment.—As previously noted, lymph-node tuberculosis is more common than all the other contagious diseases of childhood. The fact therefore that it is so prevalent and that it is so insidious should make the physician very quick to suspect its presence, and to institute proper treatment long before the serious types of this disease announce themselves in a form that places the child beyond the reach of curative treatment, and also before such diseases as measles, pertussis, influenza, and bronchopneumonia make the diagnosis by developing a curative into a much more serious form of tuberculosis. The principles which underlie the successful treatment of active tuberculosis in children are very much the same as those above outlined under Prophylactic Treatment. The child, if of school age, must stop school and live an outdoor life in a moderately bracing climate that will give him the purest air, the most sunshine, and the most equable temperature. These axiomatic facts mean that the individual child, within the limitations of the circumstances surrounding it, should have as much country life as

possible either at home or in a more suitable climate. Southern California and portions of South Carolina, Georgia, Florida, Texas, and New Mexico offer suitable winter climates, while the Adirondacks and Colorado offer satisfactory summer climates. The advantages of sanatoria in the treatment of tuberculosis in childhood are nothing like as great as they are in the adult. The sanatorium treatment in children would be of advantage to those who have not the means to take proper care of themselves at home or to take advantage of a change of climate when the conditions demand it; such sanatoria would have to be furnished by the state. The facilities for sanatorium treatment among the poor are very limited; the most that can be done for these children at the present time is to send them for short stays to "Fresh Air Farms," and other like excursions into the country. The tuberculosis dispensaries as now organized in large cities are of great value in the treatment and prevention of tuberculosis among the poor. These dispensaries, with their doctors and visiting nurses, keep in touch with their patients and, by coöperation with "fresh air" and other organized charities, are able, in a limited way, to give them better air and better food.

While the climatic treatment of tuberculous children is in selected cases of the greatest possible value, yet the fact remains that the vast majority of tuberculous children must be treated *at home,* if not for the whole, at least the greater portion of the year. It is encouraging therefore to note that the home treatment of tuberculous children among the well-to-do and middle classes is almost, or quite, as successful as any climatic or sanatorium treatment could be. The home offers many advantages, especially in the way of proper food, quiet surroundings, well-ventilated or open-air sleeping apartments, and protection from other contagions. It may require that the family remove to the suburbs of the city in which they live and there give the child an out-door life with proper food and wholesome surroundings. The fresh-air treatment of tuberculosis is, as a rule, more satisfactorily carried out in the home than it is at a summer or winter resort to which the family have flown with inadequate means to provide for themselves and their sick child the proper sanitary surroundings. If the tuberculous process is active enough to produce fever, the home is by far the most satisfactory place for the treatment of such cases. These children require rest in bed for all or at least a greater portion of the day, and they must have what all tuberculous patients demand, fresh air, sunshine, and proper food. These conditions can best be complied with at home, except among the very poor, and they can be cared for in the *fresh-air wards* of our public hospitals.

Fresh-air Treatment.—In properly carrying out this treatment the child should be required to sleep out of doors; this can be accomplished in the great majority of cases. My experience with the out-door treatment of tuberculosis, and many other diseases, has been in and around Cincinnati, Ohio; the winter climate here is like that of New York, Philadelphia and St. Louis, cold, damp, and unwholesome. From the

latter part of December to the latter part of March the climate is most variable, rains, snows, high winds, zero temperature, and thawing weather may follow each other in rapid succession, and influenza, and catarrhal diseases prevail. Yet even in this climate very remarkable curative results can be obtained from sleeping out of doors throughout the winter months. I have found that children, both sick and well, when they have once been trained to it, prefer porches and verandas to indoor sleeping apartments. During the extreme cold weather of winter, special sleeping garments and extra bed clothing are necessary, and the porch may be supplied with canvas drop curtains, one or more of which may be let down on stormy nights. Sleeping in rooms with wide open windows, while not as good, is a fair substitute for out-door sleeping apartments. The value of this fresh-air treatment for both sick and well children can scarcely be overestimated. After spending the best part of the day breathing the impure and germ-laden air of the schoolroom, it is a crime to shut children up in close and ill-ventilated sleeping apartments for the night. These are the conditions that aggravate tuberculosis and promote the spread of influenza and other contagions. Sleeping out of doors the year around is the most potent single measure we have for the cure of tuberculosis, and it is much more effective in the chronic glandular tuberculosis of childhood than it is at any other age or in any other form. In fact, this form of tuberculosis, between the ages of four and ten, yields almost specifically to the fresh-air treatment.

Dietetic Treatment.—In children this is almost as important as the fresh-air treatment and can be carried out nowhere so well as at home. The nutritional problems of the tuberculous child must be carefully studied by the physician, and a diet containing food easily within the range of his digestive capacity should be carefully prescribed. Proper food at regular intervals must be given over a long period of time. In the average child this food should be made up largely of milk, eggs, meat, and cereals, but the age and digestive capacity of the individual child must guide the physician in his selection of the diet. One fact should be firmly impressed upon the physician's mind, and that is that he can never cure a case of tuberculosis unless he is able to successfully solve the nutritional problems of the child. It is only the well-nourished child, with good digestion, continuously fed upon proper food, that finally gets well of tuberculosis. The younger the child the more important is the dietetic treatment and the less probable that a change of climate will be of greater curative value than careful dietetic home treatment. It is, of course, possible for the well-to-do to combine home life with change of climate. When this is possible we have the ideal conditions for the proper treatment of tuberculosis in children. It has not been my purpose here to undervalue the great curative power of a suitable climate in the cure of tuberculosis in children, but only to call attention to the fact that so much stress has been laid upon this factor that many children suffering from tuberculosis are carried away from good homes to a boarding-

house or hotel life that does not offer as favorable opportunities for the treatment of this disease as they left behind them. The successful treatment of tuberculous children must for the most part be carried out at home, and the climatic treatment is only a valuable adjunct, which is to be prescribed to meet the needs of the individual child.

Rest and Exercise.—Rest and exercise should be as carefully prescribed to meet the needs of the patient as any of the other measures adopted for the cure of tuberculosis. All acute exacerbations of this disease, especially those associated with fever, loss of weight, and a weak and rapid pulse, should be treated by rest rather than exercise. Whether the child shall rest in bed, with wide-open windows, or on a lounge, or in a comfortable chair, will depend upon the severity of the acute symptoms and upon its individual idiosyncrasies, the object being to give the child bodily rest without producing nervous irritability by the confinement which rest imposes. As it convalesces from acute symptoms, moderate exercise out of doors, under careful supervision, should be prescribed. In the more chronic forms of the disease, where the acute symptoms are in abeyance, more active exercise is of value. But at all times one should be careful to note that the prescribed exercise should not be of such a character or so long continued that it will produce undue fatigue or be followed by a rise of temperature. With these restrictions, out-of-door exercise is a most valuable adjunct in the treatment of tuberculosis in children. Swedish movements, general massage, mild gymnastics, and respiratory exercises are of value in individual cases, especially where there are poor chest development and general malnutrition, combined with rapid heart action and rise of temperature on moderate out-door exercise.

Medical Treatment.—While fresh air and proper diet are recognized as the all-important measures in the treatment of tuberculosis in childhood, it is my belief that the administration of drugs is a very important adjunct to this treatment and that in recent years too little attention has been paid to this phase of the subject. In administering drugs, it is an axiomatic fact that all medicines which upset the digestion, interfere with the appetite, or disturb the normal nutritional processes of the infant and child, do more harm than good, and should therefore be very carefully avoided. Creosote, guaiacol, and their derivatives have long been recognized as valuable remedies in the treatment of tuberculosis. But these remedies, when given by the mouth to children, commonly do more harm than good. For this reason I have for the last fourteen years preferred to administer them by inunction.[1] Guaiacol is especially suitable to this form of administration. The prescription introduced by me, many years ago, is as follows:

℞ Guaiacol ..... ʒ i
Lanolin (anhydrous) ..... ʒ i
Sig. Level teaspoonful externally once or twice a day.

The technique of the application of this ointment is as follows: The

[1] *American Journal of the Medical Sciences*, January, 1909.

skin of the chest and abdomen are carefully washed with soap and warm water, and, after thoroughly drying the surface, one drachm of the ointment is carefully and gently rubbed into the skin of the chest and upper part of the abdomen; the inunction should be continued from five to ten minutes. In this way the guaiacol can be introduced into the lymph and blood channels of the child and, passing through these circulating media, can be found in the urine within two hours after the application. Guaiacol administered in this way is perhaps the best lymphatic antiseptic which we possess, and there can be no doubt but that when it is thus administered we get the full medicinal and constitutional effects of the drug without disturbing the digestive organs and without interfering with the healthful nutritional processes of the body. I have arrived at these conclusions from the use of this drug, over a long period of time, in a very large number of cases. In infancy the ordinary dose is one drachm of the ointment applied once a day; in childhood the same dose twice a day. My clinical experience with the use of guaiacol in this way leads me to the belief that it is of decided value as a therapeutic measure, not only in all the chronic forms of this disease but also in the acute processes for controlling the fever, cough and nervous symptoms.

Cod-liver oil is one of the most valuable remedies we possess, especially in the treatment of the chronic forms of tuberculosis in children. It is also of great value as a prophylactic measure in the latent tuberculosis of childhood, and in improving the nutrition and increasing the powers of resistance in delicate children having a family history of tuberculosis. Cod-liver oil gives the best results when administered after meals, either in the form of pure oil or combined with one of the malt extracts, or made into a palatable emulsion; my preference is for the combination with one of the malt extracts. The individual idiosyncrasies of the patient, however, must decide not only the form in which the oil is to be given but also as to whether it should be given at all. Fresh syrup of the iodid of iron, combined with some palatable vehicle, such as a liquid diastase or essence of pepsin, is a very valuable remedy, especially in the chronic forms of lymph-node tuberculosis in older children. There is no doubt as to the value of iodin in this form of tuberculosis, and, if desirable, it may be administered by inunction, combining 5 or 10 per cent. of iodin with anhydrous lanolin. Inunctions of this ointment are of special value in superficial lymph-node tuberculosis of the cervical lymphatics; when applied in this way the remedy is quickly absorbed and appears in the urine within two hours after it is given. In the administration, however, of iodin in the chronic tuberculosis of childhood, I much prefer one of the following prescriptions:

℞ Iodonucleoids ..... grs. xl
Ferri carb. sach..... ʒ iss
M. ft. chart No. 30
Sig. One powder in half teaspoonful of malt ext. after meals. For a child 6 years of age.

℞ Comp. syrup hypophos... ℥ i
Syrupi hydriodic acidi.... ℥ i
Liquid diastase ..... ℥ ii
Teaspoonful after eating. For child 6 years of age.

It is my belief from a clinical experience extending over a number of years that these two prescriptions are of signal value in the treatment of the chronic tuberculosis of childhood. Their continuous use influences favorably the nutritional conditions of the child and slowly and gradually improves the anemia, which is such a constant symptom.

Arsenic given in the form of Fowler's solution combined with a suitable vehicle is a remedy that exercises a favorable influence on nutritional processes and improves the blood state of older children suffering from chronic forms of tuberculosis.

Tuberculin is of comparatively little value in the treatment of tuberculosis in young children. In older children, however, it may be used in subacute or chronic cases in the same manner and with the same favorable results as in the adult. In cases that are progressing favorably the injection of minute doses of tuberculin at considerable intervals lights up the latent foci and brings about a more rapid and more complete eradication of this disease. (See Vaccine Therapy.)

*Cough.*—The paroxysmal cough which is so common in the chronic tuberculosis of childhood should be treated by the bromides and tincture of belladonna combined with some suitable vehicle, such as the essence of pepsin, great care being taken not to disturb the child's digestive organs with these remedies. Chloral is also a remedy of value when the cough is unusually troublesome, but opiates are very rarely indicated. It is best to use these cough sedatives only at night, and in those cases only in which there is such an acute exacerbation of this symptom that the child's rest is very much disturbed.

*Diarrhea,* especially in older children, is best combated by small doses of oxid of zinc, one-fourth to one-half grain, combined with subnitrate of bismuth. Zinc oxid is a remedy of great value in controlling the diarrhea in intestinal and mesenteric lymph-node tuberculosis.

*Fever.*—While rest is the most important agent in the control of fever, medical remedies may be indicated. Among these may be mentioned guaiacol by inunction, phenacetin, aspirin, and pyramidon.

The treatment of tuberculosis as above outlined applies to all forms of tuberculosis in children which offer the hope of a favorable termination. It is, however, especially applicable to the great group of cases classed under chronic lymph-node and chronic bone and joint tuberculosis. It remains, therefore, only to call attention to the additional treatment which may be necessary in the special types of tuberculosis occurring at different ages in the life of the child.

Cervical Lymph-node Tuberculosis.—Because of the fact that this form is not infrequently the only tuberculosis in the body and because disease of these glands does not ordinarily imply bronchial lymph-node tuberculosis, and especially because the diseased tissues are so accessible to the surgeon's knife, it is to be considered, especially in aggravated cases, a disease in which surgical interference offers the quickest and safest means of recovery. Tuberculous cervical lymph nodes which do

not yield to the general treatment for this condition should therefore be removed by careful dissection. There is some danger that this operation, if carelessly performed, may inoculate neighboring tissues and may even produce a general tuberculosis. Following the removal of the glands the long-continued application of the general principles for the treatment of chronic tuberculosis is necessary to produce a satisfactory convalescence, and careful attention to any diseased condition that may be present in the throat, pharynx, or nose is necessary to prevent a return of this condition.

General Miliary Tuberculosis.—General miliary tuberculosis, whether it occurs in infancy or in older children, is a fatal disease, and the treatment, therefore, is to be symptomatic, always applying the principles above outlined for the general treatment of tuberculosis, in the hope that there may be a mistake in the diagnosis. Fresh air, proper food, guaiacol by inunction and the treatment of special symptoms should be carefully observed until a fatal termination is evident.

Tuberculous Bronchopneumonia.—Tuberculous bronchopneumonia, at whatever age it may occur, should be treated by rest in bed, fresh air, proper food, guaiacol inunctions, the inhalation of oxygen and warm baths. Special symptoms, such as high fever, irritable cough and gastro-intestinal complications, should be dealt with in the manner outlined under the treatment of ordinary bronchopneumonia.

Tuberculosis of the Lungs in Older Children.—This is to be treated as phthisis in the adult. A quiet out-of-door life with proper food and all the measures previously noted in the treatment of chronic tuberculosis are to be utilized. Rest in bed and suitable climatic treatment are more urgently demanded in this form of tuberculosis than in any other. High and dry air in a moderately bracing and equable climate is of value. The symptomatic treatment is the same as in the adult; the cough, fever, night sweats, and other troublesome symptoms are to be treated as in the adult.

Tuberculous Peritonitis.—Tuberculous peritonitis demands rest, fresh air, a most carefully selected diet looking to the correction of the intestinal complications, guaiacol inunctions over the abdomen, and the careful carrying out of the general principles above outlined for the treatment of chronic tuberculosis. The treatment of this form should look to the correction of the intestinal complications; carbonate of guaiacol, in from 3- to 5-grain doses, may be administered; diarrhea may be treated by bismuth, and constipation by enemata. Abdominal pain may be relieved by the application of heat, and sometimes small doses of paregoric may be necessary. Following or alternating with the inunctions of guaiacol one may employ inunctions of unguentum Credé, which is a remedy of value in many of these cases. If, however, the disease fails to yield to this treatment, surgical measures may be resorted to. Laparotomy with free drainage of the peritoneal cavity is commonly followed by improvement which not infrequently continues to a final recovery.

Tuberculosis of Bones and Joints.—This is a surgical condition,

the special treatment of which is outlined in works on general and orthopedic surgery. The general treatment above outlined should accompany the surgical treatment.

## CHAPTER XLV

### ACUTE ARTICULAR RHEUMATISM AND OTHER FORMS OF ARTHRITIS

### ACUTE ARTICULAR RHEUMATISM

Rheumatism is a general febrile disease of infectious origin, its chief manifestations being non-suppurative polyarthritis, acute inflammatory disease of the heart, and chorea. One or all of these manifestations may be present in the same case; any one may take precedence in the order of their development, but most commonly the arthritis precedes the heart disease and the chorea. These latter syndromes are elsewhere considered. Acute articular rheumatism is the term generally used to describe the polyarthritis and its associated symptoms produced by the infectious agent of rheumatism. It is most important that this broad view of the nature of rheumatism be kept in mind, since the articular manifestations in early life are sometimes so slight that it will be altogether overlooked, unless the general character of the disease is recognized and the importance of other symptoms taken into consideration in making the diagnosis. Rheumatism in childhood is not simply an arthritis; it is a general infection in which the arthritis may or may not play the most important rôle. For this broader view of the nature of rheumatism we are largely indebted to Cheadle, Barlow and their followers.

**Etiology.**—Rheumatism is generally recognized as an acute infection, but as yet the specific microorganism which produces this disease has not been positively determined. It occurs, as a rule, sporadically, but may also appear in epidemic form. It is believed that the contagion usually enters the body through the lymphatic ring of which the tonsils and adenoids are a part, and in doing so may produce acute inflammation of this lymphatic tissue. Heredity is an important predisposing factor. There is no doubt but that the members of certain families are predisposed to rheumatism. This hereditary taint in many instances is related to the gouty diathesis. The individual may inherit arthritism, or a susceptibility to inflammations of serous membranes, and by reason of this inheritance offer but feeble resistance to the rheumatic poison. Rheumatism is more common in cold, moist climates and is more frequently seen during the spring of the year. Exposure to damp cold has long been recognized as an exciting factor. Rheumatism is extremely rare under two years of age, uncommon under five, but between the seventh and fifteenth year of life it is almost as frequently observed as it is in adult life. It is slightly more common in girls than in boys.

**Symptomatology.**—In children over twelve years of age the onset is commonly sudden, as it is in the adult. The disease is ushered in with chilly sensations, followed by a rapid rise in temperature which may reach 104° or 105° F. This is accompanied by a sharp inflammation of one of the ankle or knee joints. The swelling, redness, and pain rapidly increase until the joint becomes extremely painful to motion and exquisitely sensitive to touch. These symptoms are associated with an acid perspiration, and the disease quickly spreads to other joints, producing the typical rheumatic polyarthritis seen in the adult. The patient, prostrate and helpless, cries with pain when the inflamed joints are moved or touched. This is the picture seen in the adult, and sometimes in the older child, but it is in strange contrast to the clinical picture of acute articular rheumatism in the young child, where the arthritis may be so slight as to almost escape attention, and where in many instances the child remains upon its feet until attention is called to its slightly tender joints and its slight elevation of temperature, by the development of acute endocarditis or some other manifestation of the rheumatic poison. Between these two extreme clinical pictures the severity of the disease may vary; on the whole, however, it should be remembered that in the child it is less violent in its onset and much milder in its arthritis, and that cardiac manifestations are much more common and severe than they are in the adult. The younger the child the milder the joint symptoms is a rule which may have its exceptions. In the average case prodromal symptoms are present, such as anorexia, languor, catarrhal sore throat, abdominal pain, and slight fever. In these prodromes there is nothing distinctive and the nature of the disease is not suspected until careful examination reveals tender and slightly swollen joints. Rheumatic polyarthritis is not so widespread in the child as it is in the adult. It most commonly occurs in the knee, ankle, wrist and elbow joints, and later may spread to the fingers, toes, hips and vertebra. Barlow called attention to the frequency with which the hip joint is involved in childhood. The acute inflammation in a joint rarely lasts longer than one or two days, and usually not more than two joints are acutely inflamed at the same time. The disease, spreading from joint to joint, may prolong the fever and arthritis from one to three weeks. Muscular pain and tenderness are common symptoms of rheumatism in childhood; they are due to inflammation of the fascia, and are usually more marked near the joints; many of the "growing pains" are due to this cause. Rheumatic wry-neck is not infrequent; it is caused by tonic contractions of the sternocleidomastoid, which last two or three days and are not intermittent, as they are in malarial wry-neck. This symptom is associated with some pain and marked tenderness of the muscle. Rheumatic nodules are small, fibrous nodes, varying in size from a bird-shot to a buck-shot, located near the joints and along the tendons. They are found especially about the wrists, elbows, knees, knuckles, and vertebra. They may be felt more readily than seen. When present, however, they are easily brought out by stretching the skin over these joints. In

England these nodules are common and are classed among the valuable diagnostic signs of rheumatism in the child. In America, however, they are comparatively rare. Tonsillitis is a very common rheumatic manifestation. It occurs, as a rule, as one of the initial symptoms, especially in second and third attacks of this disease. The anemia caused by rheumatism is well marked and progressive. There are few diseases that produce so great a destruction of hemoglobin and red blood corpuscles in the same length of time. Neurotic disorders are among the late manifestations; they are perhaps due to the anemia and may continue for many weeks or even months after convalescence is established. The rheumatic child is highly excitable, irritable, sleeps restlessly and may suffer from night-terrors, incontinence of urine, habit spasm, and other neurotic disorders. The acid perspiration, so common in the adult, is not present in the child.

Heart disease occurs in more than half of the cases; endocarditis is very common; pericarditis and myocarditis occur less frequently. These conditions are described in another chapter, but the facts should here be emphasized that disease of the heart occurs much more frequently as a rheumatic manifestation in the child than it does in the adult, and that the frequency and severity of the cardiac disease are in no way related to the severity of the arthritic symptoms. A case of rheumatism with little or no arthritis may develop a fatal heart disease. It is due, therefore, to the frequency and severity of the cardiac lesions that rheumatism in childhood is such a serious disease. Chorea is a common manifestation of the rheumatic poison. This syndrome may precede or follow either the arthritis or the heart disease. Pleurisy and iritis are rare manifestations of the rheumatic poison. Various skin eruptions may occur; the most important of these are purpura, erythema multiforme, and erythema nodosum.

**Diagnosis.**—If the clinical picture of this disease, as it is presented in childhood, be ever kept in mind, there should be little difficulty in making an early, accurate diagnosis. The certainty with which the salicylates modify or control the symptoms may, in doubtful cases, be an important aid to diagnosis. An acute syndrome resembling rheumatism that is not in any way influenced by the salicylates is, as a rule, not rheumatic. Again it should be remembered that rheumatism is extremely rare during the first two years of life, and is uncommon before the fifth year. A syndrome presenting joint symptoms resembling rheumatism in the adult, occurring in a child under three years of age, is almost without exception not rheumatic. Scurvy is the condition most commonly mistaken for rheumatism in infancy, but this is simply because the physician has in mind the adult type of rheumatism and has out of mind the syndrome of scurvy (see Scurvy). Syphilitic arthritis is an afebrile condition which should easily be excluded by the absence of other syphilitic symptoms. Septic arthritis, which occurs as a common manifestation of septicemia and septicopyemia, is frequently mistaken for rheumatism in childhood; the following clinical characteristics, however, should differentiate it from this disease. It follows some acute infection such as influenza, scarlet fever,

diphtheria, gonorrhea or pneumonia; the joints are acutely and sometimes very violently inflamed, and pus formation is common; it may run a more or less chronic course, the fever is septic in type, the joint symptoms are uninfluenced by salicylates, a blood examination shows well-marked leukocytosis, and the pus, aspirated from the joint, may determine the specific organism producing the inflammation. Acute osteomyelitis may be mistaken for rheumatism, but here also the high fever, marked constitutional symptoms, and pronounced swelling and tenderness which occur near but not in the joint should make the diagnosis plain.

**Prognosis.**—The prognosis, so far as the joint symptoms are concerned, is good. The arthritis quickly disappears, leaving the joints entirely free from pain and tenderness, and recurring attacks do not, except in rare instances, produce the chronic thickening and tenderness of the joints so frequently seen in the adult. The cardiac lesions, however, which are so commonly caused by rheumatism, are always serious and sometimes fatal.

**Treatment.**—With the ONSET of symptoms indicating an attack of rheumatism, the patient should be put to bed in a well-ventilated room, the temperature of which should be kept between 65° and 70° F. throughout the attack. As a rule it is advisable to keep the patient in bed for one or two weeks after all acute symptoms have disappeared. This commonly covers a period of four or five weeks. Rest in bed modifies the severity of the attack, diminishes the dangers of cardiac complications, insures a more satisfactory convalescence and prevents relapses. The diet throughout the acute attack should be milk, bread and cereals, and with the beginning of convalescence chicken and mutton broths thickened with cereals, purées of vegetables, and eggs may be allowed. When the joints are acutely inflamed they should be incased in thick layers of cotton-wool, wrapped with bandages, and immobilized by light splints outside the cotton-wool dressing. They may also be protected from the weight of the bed-clothing by shields.

MEDICAL TREATMENT.—This should be begun with a dose of one or two grains of calomel followed by Rochelle salts, and throughout the attack the bowels are to be kept open with sodium phosphate, sodium sulphate or some other saline laxative. Immediately following the preliminary cathartic, the salicylate treatment should be begun. In the great majority of cases the wintergreen salicylate of soda combined with the bicarbonate of soda will give the best results. But other salicylates such as aspirin and salol may be used.

℞ Sodii salicylatis (wintergreen) ..... ʒ i
Sodii bicarbonatis ..... ʒ i
Syrupi aurantii ..... ʒ iv
Aquæ menth pip ..... ad ʒ ii
Sig. Teaspoonful every four hours for a child four or five years of age.

There is no difference of opinion as to the value of salicylates in these cases. The only objection to their continuous administration is that it

may nauseate and otherwise disturb the digestive organs; to avoid this the vehicle carrying them should be carefully selected and changed from time to time if necessary. The action of the salicylates in rheumatism is to a certain extent specific; they reduce the fever, lessen the pain, and perhaps shorten the attack. It is also believed that both the alkalies and salicylates diminish the danger of cardiac complications. As the acute symptoms come under control, the dose of the salicylate is to be diminished one-half, and continued until the fever and arthritis have entirely disappeared; but the alkaline treatment is to be continued for weeks after the child is convalescent. If the anemia of this disease persists for two or three weeks after the acute symptoms have disappeared, some form of organic iron and of arsenic combined with malt may be given after meals. Cod-liver oil may also be a valuable tonic. If heart complications appear during the acute attack an ice-bag is to be intermittently applied over the cardiac region, absolute quiet insisted upon, and this complication is to be further treated as outlined in the section on The Heart.

Treatment of the Interval.—If the season be winter the child should be sent to a warm, dry climate, to complete its convalescence. Out-door life in an equable, warm, dry climate will quickly restore the child's health and strength. On its return home in the spring of the year, the throat and nose should be carefully examined, and, if necessary, diseased tonsils, adenoids and other growths should be removed, so that during the summer the child's throat and upper respiratory passages can be gotten into a condition to resist the common catarrhal conditions so prevalent during the winter months. During all of this time constipation and overfeeding are to be carefully avoided. It is most important that the child should have sufficient food of proper character to serve nutritional purposes, but in many of these children it will be found that they are taking from one-half to one-third more food than is actually necessary. The caloric value of the child's food should therefore be determined, that one may be sure that he is not being overfed and thus overtaxing the excretory organs. During all of this time he should live on a simple general diet, composed of vegetables, cereals, bread, meats of all kinds, eggs, cooked fruits and especially milk, avoiding sweets, tea, coffee, strong beef broth and raw food of all kinds. No medication perhaps is necessary, except an occasional cathartic or a course of bicarbonate of soda, or some other alkali, if the urine becomes hyperacid and the child becomes nervous and irritable. The underclothing should be of wool, and this should only be discarded for cotton underwear during the hot summer months. These children should be kept under medical supervision for years, and during all of this time protected from damp, cold weather, and yet be out-of-doors as much as possible; the best results are obtained, therefore, in changing the climate with the season so that they may live as much as possible out-of-doors in a warm, dry climate. It will not be possible to furnish such ideal conditions for all of our patients; most of them must be treated at home, but in the home treatment the same principles must

be carried out and the physician's judgment may sometimes be taxed to determine in an individual case whether an indoor life will do more harm than exposure to the damp, cold weather, which is so prevalent during the winter months in the temperate zone.

## OTHER FORMS OF ARTHRITIS

In addition to the arthritis which occurs in tuberculosis, gonorrhea, syphilis, scurvy, rickets, purpura rheumatica, and other hemorrhagic diseases, there are other forms which may be confused with true rheumatism and which have been described under the following names: Infectious Arthritis, Chronic Villous Arthritis, Chronic Rheumatoid Arthritis.

### INFECTIOUS ARTHRITIS

Infectious arthritis is an acute pyogenic infection involving one or more joints.

**Etiology.**—It commonly occurs as a symptom or complication of the acute infections, such as tonsillitis, scarlet fever, influenza, diphtheria, pneumonia, cerebrospinal meningitis, and septicopyemia. It may be produced by a variety of microorganisms, the most common of which are streptococci, staphylococci and pneumococci.

**Symptomatology.**—The joint or joints involved become swollen, red, tender, and fluctuation sooner or later develops. The aspirating needle reveals the character of the fluid, and from the fluid thus obtained the specific organisms producing the inflammation may be isolated. The fever in these cases is irregular or septic in type; the pain and tenderness of the joints and the fever are uninfluenced by salicylates, and the blood shows a marked leukocytosis. Infectious arthritis may be associated with or may follow osteomyelitis. In such cases the constitutional symptoms are much more pronounced, there is evidence of general sepsis and the swelling and tenderness extend from the joints into the bones.

**Treatment.**—This is a surgical condition and operative measures are usually necessary for its relief. Following the opening and draining of the infected joints, if convalescence does not readily set in, autogenous vaccines should be tried. In most cases recovery is followed by more or less complete ankylosis. Later when all inflammation has subsided massage and active and passive motion of the joint may be resorted to, in the hope of partially restoring its function.

### CHRONIC VILLOUS ARTHRITIS

Chronic villous arthritis is a low inflammatory affection of the synovial membranes, which results in hypertrophy of the villi of the joint surfaces; one or more joints may be involved. The etiological factors of this condition are unknown, but it is not uncommonly associated with other joint diseases.

**Symptomatology.**—There is little or no fever, the disease runs a very

chronic course, the joints involved gradually increase in size and assume a more or less waxy appearance; they are slightly tender, and after a time become more or less ankylosed and remain chronically enlarged. This condition may be mistaken for a tuberculous arthritis, but the absence of other signs and symptoms of tuberculosis, together with an X-ray picture showing no bone changes in the joints involved, should clear the diagnosis.

## CHRONIC RHEUMATOID ARTHRITIS

### (*Arthritis Deformans, Still's Disease*)

Under the above names are described a group of comparatively rare arthritides which occur in children, involving, as a rule, many joints, running a very chronic course and leaving the joints more or less disabled.

**Etiology.**—This condition may be associated with chronic intestinal intoxication, some defect in metabolism or some more or less obscure infection.

**Symptomatology.**—The onset may be sudden or it may be gradual, but in all of these cases in the beginning there is fever, mild or severe, with more or less soreness, pain and swelling of the joints of both the upper and lower extremities. After a time the acute inflammatory symptoms in the joints gradually subside, leaving them more or less deformed or disabled. In some cases there are repeated attacks of acute inflammation of the joints marked by increased swelling, tenderness, and perhaps by a slight rise of temperature. With these repeated attacks the joints are left more and more deformed and disabled. In one group known as Hypertrophic Arthritis the condition resembles arthritis deformans; in these cases the bones about the joint hypertrophy, exostoses form, a marked nodular deformity of the joint results, and its function is gradually lost. In another group, spoken of as Atrophic Arthritis, the tissues about the joint atrophy, and ankylosis occurs from the inflammatory adhesions of the joint surfaces; in these cases the nodular deformities about the joint are not so great. In a third group, Still's Disease, there is associated with the rheumatoid arthritis enlargement of the lymph nodes, spleen, and sometimes the liver, and marked leukocytosis is present; the lymph nodes especially enlarged are the axillary, epitrochlear and posterior cervical.

**Prognosis.**—The prognosis so far as life is concerned is good, but the majority of these cases become crippled and deformed. A certain percentage, however, especially those associated with chronic intestinal intoxication, may be greatly benefited and a small percentage entirely recover.

**Treatment.**—If the underlying intoxication can be discovered, treatment should be directed toward its removal. It is most important in all cases to give attention to the gastrointestinal canal. Careful feeding

to suit the age and digestive capacity of the individual and fresh air, night and day, are our most important remedial measures. During the acute inflammatory stage of the arthritis the joints should be fixed and carefully protected by appropriate dressings. After the acute stage has disappeared and all tenderness and evidences of inflammation in the joints have subsided, massage, passive movements, and hydrotherapy may be of value in partially restoring the lost motion.

# SECTION VII

## *DISEASES OF THE RESPIRATORY SYSTEM*

The nasal cavity of the young child is much smaller than that of the adult. In the newborn it is so narrow that slight swelling of the mucous membrane may cause its occlusion. The accessory sinuses of the nose are but poorly developed, so that one rarely sees in the infant and very young child infections of the ethmoidal and frontal sinuses. On the other hand, the Eustachian tube is unusually patulous in the young child, so that inflammatory processes of the pharynx are much more readily communicated to the middle ear. The tear ducts in the infant and child are also more patulous, and more readily carry inflammation from the nose to the eye. Catarrhal inflammations, therefore, of the ear and the conjunctiva are much more common complications of the catarrhal diseases of the respiratory passages in the young child than they are in the adult. The thorax is more cylindrical than in the adult, and the ribs are soft and flexible, being composed largely of cartilaginous tissue. This flexibility makes it possible, when there is difficulty in getting air into the lungs, for the diaphragm, by reason of its forceful contraction, to cause the chest to sink in and produce the peripneumonic groove where the diaphragm is attached to the chest wall. This peripneumonic inspiratory recession of the chest is one of the characteristic signs of dyspnea in the young infant. The softness and pliability of the ribs in early infancy is more marked in rachitic infants. Children of this type, when subjected to more or less inspiratory obstruction over a long period of time, may have chronic deformities of the chest, such as pigeonbreast and depressions of the chest wall about the lower end of the sternum.

The respiratory rhythm, like the heart rhythm of the young infant, may be very irregular without indicating a pathological condition. The frequency of respirations, like the pulse rate of the infant, may be affected by insignificant causes, and varies greatly within normal limits. Excitement, anger, slight toxic conditions, and even reflex irritations caused by pain in the intestinal canal, the ear, or elsewhere may produce a marked acceleration of the respiratory rate.

The thoracic muscles of the infant are poorly developed, but the diaphragm and abdominal muscles are well developed. The child breathes and cries with its diaphragm and abdominal muscles. The type of breathing in both sexes is largely abdominal. The costal type commences to be

manifest in the male child at about the ninth or tenth year of life. The following table shows the comparative frequency of the pulse and respirations at different periods of child life:

| | Infant. | 1st to 2d year. | 4th to 6th year. | 8th to 10th year. |
|---|---|---|---|---|
| Respiration ..... | 50-30 | 25 | 20 | 18 |
| Pulse ..... | 130-120 | 100-95 | 90 | 85 |
| Ratio ..... | 1 to 3 or 4 | 1 to 4 | 1 to 4½ | 1 to 4½ or 5 |

## CHAPTER XLVI

### DISEASES OF THE NASAL MUCOUS MEMBRANE

### ACUTE RHINITIS

(*Acute Coryza, Acute Nasal Catarrh*)

This is a catarrhal inflammation of the nasal mucous membrane which very commonly involves the pharynx and tonsils.

**Etiology.**—Infection is the prime and all-important cause of rhinitis. This catarrhal inflammation may be produced by a great variety of microorganisms, chief among which are the pneumococcus, streptococcus, staphylococcus, bacillus catarrhalis, and influenza bacillus. It may also be caused by the pathogenic organisms which produce measles, diphtheria, scarlet fever, and cerebrospinal meningitis; rhinitis, therefore, is a lesion producing a rather definite clinical syndrome, rather than a disease *sui generis*. While microorganisms are the essential cause of this condition, it should be remembered that the mucous membrane of the nose is normally in a condition to resist infection from most of these organisms; in fact the bacteria capable of producing a rhinitis may usually be found on the nasal mucous membranes of normal children. At times the disease occurs in epidemic form, being produced by the bacteria of epidemic grippe, but the fact that the ordinary or sporadic form is not infrequently produced by microorganisms which the normal mucous membrane of the nose is capable of resisting, shows the importance of predisposing factors. These factors may produce a trauma, a congestion, or an irritation of the nasal mucous membrane or, by acting through constitutional influences, may so reduce its resisting power that bacteria which are commonly present are enabled to start an acute catarrh and develop a well-marked rhinitis. Among these predisposing causes may be mentioned: traumas to the mucous membrane from chemical and mechanical causes, exposure to dry air in superheated apartments, or, more important than all, exposure to damp cold, unprotected by proper clothing. This latter predisposing cause is the one ordinarily spoken of by the laity as "catching cold," and there can be no question but that it is an important and com-

mon predisposing cause not only of rhinitis, but of all catarrhal diseases of the respiratory mucous membranes.

Rhinitis is most common in the cold, damp months of winter and spring, because the conditions for "catching cold" and for contracting contagion are very much better during these seasons. Mild cases of rhinitis are also very common during the dry fall months of the year, when the mucous membrane is so constantly irritated with a dust-laden atmosphere. The particles of dust may act as carriers of bacteria and as irritants to the mucous membrane, thus preparing the soil for the seed which is carried to it. Subacute or chronic diseases of the tonsils and adenoid tissues may be the cause of repeated attacks of rhinitis. Constitutional diseases, especially syphilis and tuberculosis, are so commonly associated with rhinitis that this symptom group is described as a part of the symptomatology of these two diseases. Other malnutritions which produce anemia and diminish the general resistance may be predisposing causes. Rhinitis is one of the important symptom groups of hay fever. There is a recurring form, probably due to autotoxins, which is described under Nutritional Disorders.

**Symptomatology.**—The onset is marked by an irritation of the mucous membrane of the nose, which manifests itself by redness and swelling; sneezing is a common and early symptom. As a rule there is a slight elevation of temperature associated with headache, lassitude, sleeplessness, nervousness, and more or less general discomfort. Early in the disease the nasal discharge is thin and watery, being serous in character. In a few days this discharge becomes thicker and more tenacious, being composed of heavier mucus; later it may become mucopurulent. In gonococcic, diphtheritic and scarlatinal infections the discharge is always purulent. The irritating character of the discharge in ordinary rhinitis varies greatly. Not infrequently the nasal openings and the underlying portion of the lip, over which the discharge runs, are irritated, red, excoriated and sometimes covered with dry scales, or scabs, which may partially or completely block up the nasal opening. Rhinitis is a comparatively insignificant disease, which runs a mild course, terminating in recovery in from three to five days. The great majority of the cases are fortunately of this type. In the very young infant the disease is much less commonly seen, but when it does occur it is a source of much greater anxiety to the physician, not only because it is more likely to extend through the pharynx to the bronchial mucous membrane, but also because even simple rhinitis at this age is a troublesome, sometimes serious, and rarely dangerous affection, because the swelling of the nasal mucous membrane not infrequently occludes the narrow nasal passages of the young infant, and when this occurs it may have great difficulty in sleeping, breathing, and taking food. The very young infant, not being accustomed to breathe through its mouth, may be put in a perilous position by having its nasal passages occluded; dyspnea, severe attacks of asphyxia, and, in rare instances, even death may result from nasal stenosis; this, however, is a very

rare occurrence. The taking of food in these cases is always more or less interfered with; the infant often cannot nurse either from the breast or from a bottle, since it must let go of the nipple to get its breath; it may be necessary to feed with a spoon.

*Cough.*—In simple, uncomplicated rhinitis cough is usually absent, but in the majority of cases the disease extends to the pharynx and sometimes to the larynx. Irritation in either of these regions may produce a cough, the character of which may mark the progress of the disease. The pharyngeal cough frets the infant and is sharp and irritating; as the disease progresses into the larynx, the characteristic croupy cough, later to be described, makes its appearance.

*Fever.*—Rhinitis may run its course with little or no fever; however, there is usually a slight elevation of temperature in the beginning of the disease. In the epidemic forms associated with grippe and other contagious diseases the temperature at the onset may be high; this is not due to the coryza, but to the general infection of which the coryza is a symptom. Later in the disease, as the rhinitis is running its course, apparently in a satisfactory manner, we may have a sudden and marked elevation of temperature, the fever reaching 104° or 105°F. within a few hours; this commonly means the onset of an acute otitis media. This complication demands immediate surgical attention; an early incision of the drum may save not only much suffering, but a possible mastoid infection. Earache commonly precedes the otitis media; but in some instances, especially in malnourished, tuberculous children, a discharge from the ear is the first indication of this complication.

*Pharyngitis, tonsillitis,* and *inflammation of the adenoid tissue of the pharynx* are frequently associated with coryza. Catarrhal inflammation in the nose not infrequently spreads to the eye, producing a mild, or even a pronounced, conjunctivitis; this may occur in epidemic form, and is then commonly spoken of as "pink eye." The conjunctivitis may precede the coryza, but commonly the reverse is true.

*Pseudomembranous rhinitis* should always be looked upon as diphtheritic, until it has been definitely demonstrated to be due to other causes. The safest plan in these cases is to give a dose of diphtheria antitoxin, and later determine by a bacteriological examination whether the membrane is diphtheritic or due to diplococci, streptococci, staphylococci, or other microorganisms. The bacteriological examination, in fact, in these cases is not always to be relied upon. In cases of simple rhinitis I have seen capable bacteriologists demonstrate bacilli which could not be differentiated from diphtheria bacilli, and in advanced cases of diphtheria of the nose I have seen them fail to differentiate the specific microorganism of this disease. It is wise, however, in every case of severe rhinitis, associated with marked irritation and constitutional symptoms, to make a bacteriological examination, and if diphtheria bacilli are found, membrane or no membrane, antitoxin should be given.

**Prognosis.**—The prognosis in the vast majority of cases is good; the

disease runs a short and benign course and terminates in recovery. In the diphtheritic and other pseudomembranous forms the disease may terminate fatally. The prognosis in these cases largely depends upon the treatment. During the first year of life even simple rhinitis may become a dangerous disease which occasionally terminates fatally.

*Chronic rhinitis,* as compared with its frequency in the adult, is uncommon in the infant and young child. Chronic nasal catarrh rarely supervenes upon the acute process except in tuberculous and syphilitic children, or in those who have chronic disease of the tonsils or adenoids. A relapsing, or chronic coryza in an otherwise healthy infant usually means chronic disease of the adenoid tissues of the pharynx.

**Prophylaxis.**—As rhinitis is an air-borne disease, due primarily to infection, and secondarily to causes which irritate the nasal mucous membrane, the prophylactic treatment consists in keeping the child in fresh air day and night. It should live in the open as much as possible during the day, and sleep in a well-aired room at night. Well children should be kept away from sick ones. Infants and young children should be kept out of closed street cars and places of amusement, where large numbers of people are crowded together in a close, overheated atmosphere. Recurring attacks of rhinitis may require the removal of diseased tonsils and adenoids.

**Treatment.**—Children suffering from simple rhinitis should be kept out of doors in the fresh air, away from dusty streets and roads, that they may breathe pure air, free from germs, dust, and other irritating impurities. Living indoors and breathing warm, dry, impure air aggravate the disease. Nasal injections of some mild alkaline antiseptic are of value, especially in older children; they should be given with a soft all-rubber syringe, the child's head being inclined forward with the face looking downward, and the fluid gently and slowly injected into the anterior nares in such a way that the mucous membrane of the nose and pharynx may be irrigated. If the child is not old enough to accomplish this operation without a struggle, it is better not to attempt it at all; these same antiseptic applications may be almost as effectively applied with atomizers; this is preferable to the nasal injections in young children. Inhalations of cresolin, tincture of benzoin, guaiacol and oil of turpentine may be used to advantage in steam atomizers. In every case of rhinitis, both in infants and older children, the following prescription may be instilled into the nose, four or five drops three or four times a day: Oil of eucalyptus, m. 10; menthol, gr. ½ to 1; liquid albolene enough to make an ounce. This is an effective remedy, which may be used even in the newborn. If in a given case the mucous membrane of the nose be very raw and irritated, the menthol may for a time be left out of the prescription; it is, however, an effective antiseptic, and in the dose above given is usually not irritating. In cases where there are great irritation and swelling of the mucous membrane of the nose the following prescription may be used:

℞. Cocain hydrochlorid ..... gr. i
Adrenalin sol. (1 to 1,000) ..... ʒ i
Boric acid ..... grs x
Distilled water ..... ad ℥ i

From three to five drops of this mixture may be dropped into the nose at intervals of three or four hours. It has a sedative action and tends to relieve the engorgement of the mucous membrane. An ointment of lanolin containing 1 per cent. of boracic acid is a soothing application to the external nares and upper lip when these parts are irritated and excoriated. This may be used on pledgets of cotton or gauze to remove the crusts and cleanse the external nasal canal. In desperate cases in very young infants a small, soft catheter has been recommended for introduction along the nasal canal to prevent its complete closure. In even more desperate cases tracheotomy has been resorted to to save the life of the suffocating infant. These extreme measures are very rarely necessary.

With the local treatment above recommended, a number of drugs may, in selected cases, be used internally. In older children quinin is of decided advantage, and is to be given in pill form, if possible. If the child be too young for this, one or two grains of euquinin may be given at three or four-hour intervals. In infants under eighteen months of age the following prescription may be used:

Guaiacol carb. ..... grs. xii
Salol ..... grs. xii
Sugar ..... grs. xii
Make 12 powders.
One every three or four hours.

The treatment of chronic rhinitis comprehends the use of all the remedies above mentioned, and in addition the removal of the underlying cause. This is usually some constitutional disease such as tuberculosis or syphilis, or some chronic local disease such as hypertrophied adenoids and tonsils. With the removal of these conditions, chronic and recurring rhinitis in the child usually disappear.

## EPISTAXIS

Nose-bleed is rare in young infants. It may occur during the first days of life as a symptom of syphilis or sepsis.

**Etiology.**—The exciting cause is commonly some injury to the mucous membrane of the nose produced by contusions, by foreign bodies or by other traumas. But the direct exciting causes, in the majority of instances, are of less importance than the predisposing causes, since by them the mucous membrane is put in such a condition that it bleeds from the slightest injury. Among the predisposing causes are adenoid vegetations, nasal catarrh and ulcerations of the nasal mucous membrane. In certain infectious diseases, such as typhoid fever, measles, influenza, scarlet fever and

whooping-cough, nasal hemorrhages are common. They occur also in certain constitutional diseases, such as hemophilia, purpura hemorrhagica, peliosis rheumatica and grave forms of anemia, and scurvy. Where the predisposing causes are marked, nasal hemorrhages may sometimes be produced by stooping and by violent exercise, or they may be spontaneous; that is to say, the exciting causes are not discoverable.

Hemorrhages occurring from the back part of the nose may, especially in infants, cause the blood to flow into the pharynx, where it is swallowed, and produces a dark discoloration of fecal discharges. Nasal hemorrhage unassociated with constitutional disease is rarely severe enough to produce marked anemia or general weakness. In purpura hemorrhagica, severe toxemia, and hemophilia nose-bleed may become dangerous. In the majority of cases the bleeding point may be located by an examination with a nasal speculum.

**Treatment.**—In most instances no treatment is required; the bleeding stops spontaneously after a short time; the home remedies, such as swallowing salt and cold applications to the back of the neck, occupy the attention of the family until the hemorrhage ceases. The most effective remedy for the relief of nasal hemorrhage is the injection through the nasal canal of an adrenalin solution after the clots have been removed; for this purpose the 1 to 1,000 solution may be diluted ten times and injected through the nose with a soft rubber syringe; this same solution may be applied on pieces of cotton or on strips of gauze which are pushed into the nose beyond the bleeding point. Other remedies are rarely, if ever, needed for the control of ordinary nasal hemorrhage. Bleeding from the nose may very commonly be stopped by the simple introduction of pledgets of dry cotton, without the use of medicines to contract the bleeding vessels. In cases of nasal hemorrhage associated with severe hemorrhagic constitutional diseases it may be necessary to tampon the whole nasal cavity with gauze, saturated with adrenalin solution. In recurring attacks of epistaxis the bleeding point in the nose may require cauterization in the interval between attacks.

## FOREIGN BODIES IN THE NOSE

Young children very frequently push small foreign bodies into the nose, such as buttons, grains of corn, beans, pebbles, beads, and other small objects with which they play; thus lodged in the nostrils they often remain for days or months before they are discovered. Foreign bodies in the nose produce more or less occlusion of the nasal passage, and as a result a unilateral rhinitis occurs; this may be severe enough to produce a very decided inflammatory process with ulceration. In the great majority of instances the foreign body is discovered before it has produced marked inflammation, and its removal from the nose is, as a rule, a simple process. If the child be old enough it may, by closing the opposite nostril, force the body out by blowing the nose; in most instances it can

readily be seen and pulled out with a pair of fine forceps. If its shape be such that it cannot be grasped by forceps, it can be removed by introducing a small probe which is bent very slightly at the end; this may be passed beyond and hooked over it. In some instances it may be necessary, where the swelling and inflammation are great, to relieve the sensitiveness of the mucous membrane by the application of cocain. Where the foreign body is so far back in the nasal passage that it cannot be removed in one or the other of the ways described, it may be pushed with a small, cotton-wrapped probe back into the pharynx, being careful that it does not drop into the larynx or trachea.

## CHAPTER XLVII

### DISEASES OF TONSILS

The faucial tonsils are a part of Waldeyer's lymphatic ring which extends around the pharynx, and includes the pharyngeal tonsil (adenoids) and, later in life, the lingual tonsil. These tonsils are masses of lymphoid tissue which are held together by intervening connective tissue. In the infant and young child the pharyngeal tonsil (adenoids) is most commonly affected by disease. In childhood the faucial tonsils, which are comparatively inactive during infancy, are very commonly diseased, and later in life the lingual tonsil may be a source of trouble. The faucial tonsils, whose function is more or less obscure, are filled with crypts and contain a large number of mucous glands. The irregularity of their surface, as well as their mucous coating, enables them to stand guard at the entrance to the throat and prevent microorganisms and other disease-producing factors from entering the pharynx; the microorganisms thus picked up are usually cared for in a satisfactory manner by the normal tonsil without producing disease. There can be no question but that the tonsils, especially in children, serve an important purpose in preventing contagion. The crypts very commonly contain not only mucus and particles of food, but large numbers of pathogenic microorganisms, including pneumococci, diplococci, streptococci, staphylococci, and bacilli catarrhalis, any of which are capable of setting up inflammatory processes in the mucous membrane of the respiratory passages; even when the tonsil itself becomes diseased or infected, the infection is rarely transmitted directly to internal organs. Jacobi called attention to the fact that while Waldeyer's lymphatic ring is one of the important gateways through which infections of various kinds enter the body, the entrance is not effected, as a rule, directly through the faucial tonsils, but when other portions of this lymphatic ring are affected, then there is great danger of the contagion obtaining entrance to the general lymph or blood streams, and producing thereby general constitutional diseases or localized infections of internal organs. The late Dr. Frederick Packard called attention to the fact that tonsillitis

very frequently preceded or was associated with endocarditis, rheumatism and other infections. While the normal tonsil may serve the important purpose of protecting the infant from contagious diseases of various kinds, the hypertrophied and chronically diseased tonsil, harboring in its crypts infectious microorganisms, frequently becomes a menace to the health of the child rather than a protecting agency against disease. In such tonsils repeated attacks of tonsillitis may occur from slight predisposing causes without new infection. These are the cases that are most closely associated with endocarditis, chorea, and acute rheumatism; and with successive attacks of ulcerative tonsillitis there may be repeated attacks of arthritis, endocarditis, or chorea. The lymphoid ring, of which the tonsils are a part, is in close communication not only with the retropharyngeal lymph glands, but also with the cervical lymphatics situated below the angle of the jaw, along the lines of the great vessels of the neck. In diseases of this lymphoid ring, therefore, the cervical lymph nodes below the angle of the jaw are more or less swollen; the retropharyngeal lymph nodes are more closely connected with the pharyngeal tonsil, and the cervical lymph nodes with other portions of this lymphatic ring, including the faucial tonsils. From what has been said, it is evident that tonsillitis, pharyngitis, and adenoid disease are very commonly a part of the same pathological process. In the infant and young child practically every tonsillitis is accompanied by more or less pharyngitis, although the reverse of this is not true.

## TONSILLITIS

**Acute Follicular Tonsillitis.**—Etiology.—Acute follicular tonsillitis is an infectious disease which may be produced by a number of pathogenic microorganisms, chief among which are diplococci, streptococci, staphylococci, pneumococci, micrococci catarrhalis, and influenza bacilli. It not infrequently occurs as an epidemic, spreading through families, schools, and institutions for children. It is very commonly an important part of the syndrome of some of the acute infections, such as scarlet fever, influenza, rheumatism and measles. Any of the above-named microorganisms may be held for a long time in the tonsillar crypts, until an exciting cause starts them into activity; recurring attacks of tonsillitis are usually produced in this way. "Catching cold" and traumatism are exciting causes, which can produce tonsillitis only when the contagion is present in the tonsillar crypts. In infants it is also believed that the fermenting contents of a disordered stomach may be the exciting cause. On the other hand, there can be no question but that gastrointestinal indigestion and infection are very commonly secondary to tonsillitis, the infected mucus when swallowed being the exciting cause.

Certain constitutional diseases, such as tuberculosis, the lymphatic diathesis, rheumatism, and gout, may predispose to tonsillitis.

Symptomatology.—In the infant and young child, not able to locate its pain, or point out the site of the disease, tonsillitis may be overlooked,

unless the physician adopts the rule of carefully inspecting the throat of every sick child. It usually announces itself with fever, pain, general discomfort, and in some instances with more or less marked prostration. The fever may rise as high as 102° or 104°F., and usually lasts from two to four days; during this time there may be marked irregularities of the temperature. There is nothing specific in the temperature curve, but it is important to remember that more or less fever is a symptom of every case of tonsillitis, and that if it lasts longer than five or six days there is probably some complication, such as otitis media, suppuration of lymph glands, or the spread of the inflammation from the tonsils to some other portion of the respiratory passages. The fever is accompanied by malaise, headache, backache, and sometimes is associated with a chill; chilly sensations are very common in older children. Young infants take their food badly, nurse with difficulty, and their breathing may be more or less obstructed, especially during sleep. Older children may complain of sore throat and pain in swallowing; the lymph nodes at the angle of the jaw are enlarged.

The diagnosis is made by an inspection of the throat. The tonsils are red and swollen; the neighboring mucous membranes of the pharynx and pillars of the soft palate may also be inflamed, and all of these tissues may be covered with a mucopurulent discharge. In almost every case of tonsillitis not only the parenchyma, but the glandular structures of the mucous membrane, are involved, and sooner or later small grayish-white spots are to be seen scattered over both tonsils; these may enlarge, run together, and form irregular, grayish-yellow patches, which are thin, and cling lightly to the tonsillar tissue, filling the crypts and in some instances covering the greater portion of the tonsillar mucous membrane. Between the membranous deposits, however, strips and patches of swollen and red mucous membrane may usually be seen. More rarely a pseudomembrane, croupous, but non-diphtheritic in character, may form.

**Ulceromembranous Tonsillitis.**—Ulceromembranous tonsillitis, or Vincent's angina, is a form of tonsillitis produced by the symbiotic action of Vincent's bacillus and spirillum. The bacillus is fusiform in shape, shows transverse markings, has pointed ends, and is much longer than the diphtheria bacillus. The spirillum is slender and usually has three or four whorls. Vincent's angina is much less common, runs a milder course and has fewer constitutional symptoms than ordinary follicular tonsillitis; it not infrequently involves only one tonsil. The diagnosis is made by finding the microorganisms, which are readily detected in smear preparations, and by the presence of a grayish-yellow ulcer on one or both tonsils, which usually varies from one-fourth to one-half inch in diameter, but may cover the whole tonsil. This form of tonsillitis may be associated with a membranous stomatitis of the same character.

**Cause and Prognosis.**—Follicular tonsillitis usually runs its course in from three to five days. Following the acute symptoms, there may be a rather slow convalescence covering a week or ten days, during which time the patient recovers his appetite and strength, and the tonsils grad-

ually diminish in size and resume their normal color and appearance. In Vincent's angina the disease runs a longer course, and convalescence is delayed.

The prognosis in all forms of acute tonsillitis is good; the great majority of these cases recover without complications. It should be remembered, however, that otitis media, peritonsillitis, chorea, endocarditis, and septic arthritis are dangerous complications which may possibly occur. The danger from these complications, as previously noted, is much greater in the frequently recurring attacks of acute follicular tonsillitis associated with chronic tonsillar hypertrophy.

**Differential Diagnosis.**—With the onset of every tonsillitis, the physician should be on the lookout for influenza, scarlet fever, and diphtheria. From influenza and scarlet fever acute tonsillitis is differentiated by the general symptom-complex of these diseases. From diphtheria, however, it is practically impossible in many cases to make a differential diagnosis, except by a bacteriological examination. In all cases of tonsillitis which clinically resemble diphtheria it is wise to give a dose of antitoxin without waiting for the bacteriological examination to determine the presence or absence of Klebs-Löffler bacilli. In the great majority of cases the pictures presented by true tonsillar diphtheria and acute follicular tonsillitis are fairly distinct. In diphtheria the exudation presents the appearance of a membrane covering all or part of the ulcerated tonsil; it is dark gray in color, and so closely attached that any effort at its removal produces bleeding. In addition to the large membranous patch small patches similar in character may be seen on the uvula or pharynx. This picture is very different from the widely disseminated, grayish-white, small patches seen in follicular tonsillitis, and even when these enlarge and coalesce to form larger patches of membrane the exudate thus formed is rather loosely adherent, and, as a rule, easily removed without producing hemorrhage. The differential diagnosis in difficult cases must be made first by the response of the disease to antitoxin, and second by a bacteriological examination of the throat.

## CHRONIC TONSILLAR HYPERTROPHY

This is the condition previously referred to of chronically enlarged and diseased tonsils, so frequently seen in children who have suffered from repeated attacks of tonsillitis. It is commonly associated with chronic disease of the adenoids and with more or less chronic hypertrophy of the entire lymphoid ring of the pharynx. A small percentage of these cases is due to the tubercle bacillus.

**Symptomatology.**—Patients suffering from chronic tonsillar hypertrophy commonly lack strength, are malnourished, anemic, and have poor chest development. They are restless, nervous, sleep poorly and commonly speak with an altered nasal tone, and many are mouth-breathers from complicating adenoids. The lymphatic glands at the angle of the jaw

are chronically enlarged. The diagnosis is made by an examination of the throat. The tonsils are enlarged and covered with irregular deep crypts, in which not infrequently caseous material accumulates, presenting the appearance of isolated white patches. In such cases the breath is offensive and the caseous material, when removed by a dull instrument, has the same bad odor. These are the cases in which the tonsils no longer act as safeguards against infection, but are an actual menace to the health of the child, subjecting it to the dangers of tonsillitis, middle-ear infection, endocarditis, arthritis, diphtheria, scarlet fever, and other infections. There is little doubt but that in many of these cases in which there is great chronic enlargement of the tonsils this condition acts injuriously upon the health of the child by mechanically interfering with the respiration and producing a low form of chronic toxemia. Chronic tonsillar hypertrophy may be a symptom of the lymphatic diathesis; in such cases, like the anemia, malnutrition, and lack of development, it is an expression of a general constitutional disorder.

## PERITONSILLAR ABSCESS

Peritonsillar abscess, or quinsy, is comparatively rare in infancy and young childhood. The microorganisms producing this abscess are apparently the same varieties of streptococci and staphylococci found in ordinary tonsillitis, and yet the disease not infrequently occurs in epidemic form. That is to say, in certain epidemics of tonsillitis, quinsy may be common; in others it may be a rare occurrence. Individuals who have had one attack of quinsy are much more liable to second and third attacks. The disease usually occurs on one side; it may, however, be bilateral.

**Symptomatology.**—Fever, chilly sensations and a painful sore throat mark the onset of quinsy. The pain becomes very severe, is throbbing in character, and is very much aggravated by swallowing and talking. There is great tenderness and more or less swelling and tumefaction beneath the angle of the jaw in the region of the tonsil. Difficulty is experienced in opening the mouth.

**Diagnosis.**—The diagnosis is made largely upon the fact that the pain is out of all proportion to the appearance of the throat on examination. The follicular tonsillitis, which may have been present in the beginning, has entirely disappeared, but the tonsil, and especially the supra-tonsillar tissue, remains red and edematous, and on examination with the finger, fluctuation may be found. The abscess continues to increase in size, and if not opened breaks spontaneously after several days, and discharges into the throat a quantity of pus, more or less tinged with blood. The relief which follows the evacuation of the pus is very great, and convalescence is usually rapidly established.

## TREATMENT OF DISEASES OF THE TONSILS

**Treatment of Follicular Tonsillitis.**—With the onset of acute symptoms the child should be put to bed and isolated. A liquid diet suitable to its age should be selected, not only with reference to protecting it from gastrointestinal complications, but also with the idea of throwing as little work upon the excretory organs as possible. A milk and cereal diet is to be recommended at the onset, until it is definitely determined that the tonsillitis is not the beginning of scarlet fever, diphtheria, influenza, or some other acute infection. When it has been decided that only a simple follicular tonsillitis is present, the diet in older children may be increased to suit the demands of the child. In most instances the difficulty in swallowing causes the child to refuse food. In older children ice-cream, thick gruels, milk-toast, and soft, semi-solid food are more grateful and more easily taken than milk alone. The rest-in-bed treatment should be continued as long as the child has fever and marked throat symptoms. In every case the physician should carefully examine the heart in anticipation of the possible development of acute endocarditis, and frequent urinalyses should be made, as albuminuria may occur.

Medical.—The medical treatment consists in giving quinin, sodium salicylate, aspirin, phenacetin, or salol. Quinin is a valuable remedy and should be given to all children who are old enough to take pills or capsules. In younger children, and especially in infants, quinin, because of its taste, is contraindicated. The struggle to give an infant quinin in liquid form may not only produce great nervous excitement, but may upset its stomach and cause it to refuse food and other medication. Salicylate of soda from oil of wintergreen is a valuable remedy in older children. It may be given in capsule combined with the quinin, or in solution put up with glycerin and peppermint water. For a child from six to ten years of age, two grains of quinin and three grains of salicylate of soda may be given every four to six hours. In younger children aspirin is a valuable remedy; it may be given, combined with sugar, in one-grain powders to a child two years of age; this dose may be repeated at three-hour intervals. One grain of phenacetin, one or two grains of salol, and one grain of sugar may be given as a powder to infants between the ages of one and two. This prescription is effective in protecting the gastrointestinal canal, reducing the temperature, relieving the nervous irritability, and making the infant altogether more comfortable. The above remedies are to be used during the acute stage of the disease, which lasts but two or three days; the aspirin, and especially the sodium salicylate, may be given, however, for a longer time to older children in whom there is a clear family history of gout or rheumatism. At the very onset of the disease the infant or child should be given calomel in small doses until one or two grains have been taken. This is to be followed by a dose of castor-oil, or saline laxative; the castor-oil is preferable. On the third or fourth day of the

treatment a second dose of castor-oil should be given; the oil serves the purpose of clearing the intestinal canal and preventing gastrointestinal complications; the germ-laden mucus, which is swallowed, can in no manner be so satisfactorily carried off. The care of the intestinal canal is especially important in the treatment of tonsillitis in infants under two years of age; this applies with equal force to the treatment of all catarrhal conditions of the respiratory passages. Intestinal infection and gastro-enteritis are not only troublesome, but dangerous complications, much more serious than the tonsillitis which produced them. Infants, therefore, suffering from tonsillitis should have their milk formulas reduced, and if diarrhea appears it should be treated by diet and proper medication.

Local Treatment.—In the majority of instances relief and benefit follow the application of cold to the neck. This may be applied in the form of cloths wrung out of ice-water, or by a small ice-bag wrapped in a towel, and placed under the angle of the jaw over the tonsillar region. Cold applications are of special value when the lymphatics at the angle of the jaw are enlarged, when there is a throbbing sensation in the throat with marked tenderness externally over the tonsils, and in individuals who have had repeated attacks of quinsy; the early application of cold may in these cases prevent the formation of a peritonsillar abscess. In some cases very hot applications applied to the neck give more relief than cold; in younger children and infants the hot applications are, as a rule, preferable. Older children should gargle, or use a spray of peroxid of hydrogen, diluted two or three times with water; this is indicated for twenty-four or thirty-six hours only. It is an excellent throat antiseptic, but if continued too long, as Jacobi long ago pointed out, irritates the mucous membrane. As the white patches disappear on the second or third day, the peroxid of hydrogen solution is to be changed for some mild alkaline antiseptic, which may also be used as a gargle, or with an atomizer, so as to thoroughly cleanse the throat and pharynx of the mucopurulent discharge which is present. These alkaline antiseptic solutions may be made by adding boracic acid to a physiological salt solution, or by using some of the alkaline antiseptic tablets now on the market. It may be necessary to paint the throat or use stronger or more astringent gargles and sprays. A weak solution of the tincture of chlorid of iron, one to four or five parts, may be used for painting the tonsil during convalescence. Weak iodin and silver (argyrol) solutions may also be used for swabbing the tonsil, but on the whole these stronger applications are rarely indicated in the convalescence from acute tonsillitis. They are of more value in the subacute or chronic forms of tonsillar hypertrophy.

Infants and children suffering from tonsillitis are made more comfortable by sponge and tub baths, which relieve the nervousness and reduce the temperature. During convalescence older children are benefited by such tonics as fresh air, good food, and the malt and iron preparations. The tincture of chlorid of iron and the syrup of iodid of iron are old

and time-honored remedies of value in these cases. They may be given after meals in three to five-drop doses diluted with glycerin and water.

**Treatment of Vincent's Angina.**—The treatment of Vincent's angina consists chiefly in the careful local application of caustics to the ulcerated area, such as strong nitrate of silver solutions and chromic acid.

**Treatment of Peritonsillar Abscess.**—Peritonsillar abscess, or quinsy, should be treated by opening the abscess with a guarded bistoury, and the throat, for a number of days following the incision, should be disinfected by some of the alkaline antiseptics above mentioned.

**Treatment of Chronic Tonsillar Hypertrophy.**—The treatment of chronic tonsillar hypertrophy falls within the domain of the throat specialist, rather than the general practitioner. The guillotine in the hands of an inexperienced operator may remove the greater part of the tonsils and give relief for a number of years, but if tonsillar tissue be left there will be in most instances a gradual return of the tonsillar tumor, and a second operation some years later may be necessary. For this reason the radical operation of enucleating the entire tonsil within its capsule is much to be preferred. Adenoids and other hypertrophied lymphoid tissues of the lymphoid ring of the pharynx should always be removed at the same time; this is a slight operation, which should follow the removal of the tonsils. It may be well also to note that a white, innocuous membrane, somewhat resembling diphtheria, forms over the wound produced by removing the tonsil.

## CHAPTER XLVIII

### DISEASES OF THE PHARYNX

### ADENOIDS

In 1868 Dr. William Meyer, of Copenhagen, called the attention of the medical world to the hypertrophy and disease of the lymphoid tissue, which so commonly occur in the vault and posterior and lateral walls of the nasopharynx. This lymphoid tissue is spoken of as Luschka's tonsil, or the pharyngeal tonsil, and the hypertrophy is commonly spoken of as adenoid growths. Other portions of the pharyngeal wall, however, notably its posterior surface, are rich in lymphoid tissue, and these lymphoid follicles are commonly markedly enlarged when there is any great increase in the size of the pharyngeal tonsil. These enlarged follicles, therefore, which may be readily seen on the posterior wall of the pharynx, are an important indication of the presence of adenoid growths. The faucial tonsils are also commonly diseased and hypertrophied in the presence of marked adenoid disease; this, however, is not always so, since extensive adenoid growths, almost filling the vault of the nasopharynx, may be present with little or no disease of the tonsils; this is more commonly seen un-

der three years of age. In adenoid growths the orifices of the Eustachian tubes are not infrequently surrounded by diseased adenoid tissue.

**Frequency.**—Much difference of opinion still exists as to the frequency of this disease. It has been variously estimated that from 10 to 35 per cent. of all school children between the ages of six and ten, living in cold, damp climates, such as are found in our middle and northern states, have sufficient adenoid disease to demand operative interference. It should be remembered that the pharyngeal tonsil is normal tissue, and that a moderate amount of hypertrophy may exist without producing either local or constitutional injury. The question, therefore, for the physician to decide is not whether the child has adenoids, but whether the adenoids are sufficiently enlarged or diseased to produce either a local or constitutional disturbance which injures its health.

**Etiology.**—Adenoids are especially common between the ages of four and ten, but they are not infrequent during the first year of life and may be congenital. Heredity, the lymphatic diathesis, glandular tuberculosis, and cold, damp climates are classed among the predisposing causes, but the real cause of the disease is infection. The adenoid tissue becomes more and more hypertrophied with repeated infections, and in its folds the microorganisms, capable of producing acute inflammation, are held from one acute attack to another. All the etiological factors of rhinitis become the etiological factors of adenoid growths, since repeated attacks of coryza are almost constantly associated with hypertrophy of this lymphoid tissue. Colds in the head, ordinary epidemic grippe, true influenza, measles, and all the acute infections capable of producing catarrhal disease of the mucous membrane of the nose and pharynx may be etiologically related to adenoid growths.

**Symptomatology.**—The symptoms vary greatly with the extent of the hypertrophy of the lymphoid tissue, with the severity of the inflammation, and with the associated complications. The most characteristic symptoms are recurring attacks of rhinitis, tonsillitis, pharyngitis, and laryngitis, with snoring and mouth breathing in the intervals between these attacks. Sleeping and waking, the child's mouth is partially open; this is due to partial nasal obstruction. The voice is thick, muffled, and frequently has a nasal twang. Earache and partial deafness are common. Otitis media may occur. An unexplained running of the ear, which fails to yield to ordinary treatment and which is associated with recurring attacks of pharyngitis, is almost always due to adenoid growths. Recurring attacks of epistaxis are not infrequent. Laryngitis and bronchitis very frequently follow the acute pharyngitis, which is from time to time lighted up by chronic adenoid disease. The nervous symptoms associated with adenoid disease vary greatly. In aggravated cases the child may suffer from sleeplessness, general nervous irritability, headache, night-terrors, and incontinence of urine. There can be no question but that pronounced adenoid growths, occurring in malnourished and neurotic children, may produce very pronounced reflex neuroses. In such cases I have frequently seen

night-terrors and incontinence of urine disappear when the adenoids were removed. An enlarged chain of lymph nodes behind the sternocleidomastoid muscle, when associated with the catarrhal symptoms above described, is strongly confirmatory of adenoid growths. In marked cases of adenoid disease which have existed for a long time, the facial expression of the child may strongly suggest the condition. He has a stupid, vacant look, his mouth is open, the bridge of his nose is flat, his upper lip appears thick, the nasolabial fold is obliterated, and his lower jaw protrudes in such a manner as to give the appearance of a long face which narrows toward the chin. The hard palate may show a very high arch and the upper teeth may be displaced. Not infrequently these children have narrow, poorly developed chests, and are below par in their physical development. They also have the appearance of being below normal in their mental development; this, however, is perhaps largely due to their stupid expression and to the fact that because of partial deafness, or their frequent attacks of illness, they have not had mental training in keeping with their age. The mental deficiency in these cases is not real, but merely apparent, and disappears quickly with the removal of the adenoids and the improvement of the child's general physical condition. In young infants the nasal occlusion caused by adenoid disease interferes with nursing. As the infant sleeps, its mouth and pharynx become dry, and it not infrequently awakens with a choking cry and for a time may have difficulty in getting its breath. These symptoms are more commonly due to the associated rhinitis than to the adenoid disease.

**Diagnosis.**—The diagnosis of adenoids may be suspected or even made from the above symptom group, but the extent of the disease and frequently its existence can only be definitely determined by digital examination of the nasopharynx. For this examination the physician stands behind the patient and holds the child's head firmly with his left arm; the finger of his right hand is quickly introduced back of the soft palate, high up into the nasopharynx; there the location, the extent, and the character of the adenoid mass may be felt. The adenoid tissue is usually soft and friable; the examining finger therefore comes away bloody. In other instances, small, hard adenoid masses are located, which do not break down readily. During this examination, which requires but a few seconds, the child's mouth is to be held open with a mouth-gag, or with the fingers of the left hand, pushing in the cheek between the molar teeth, otherwise the finger of the right hand, which is making the examination, may be bitten or otherwise injured.

**Treatment.**—The medical treatment of acute adenoid inflammation is in every way similar to the treatment of rhinitis, which has been already given in detail. The treatment of acute catarrhal inflammations of the tonsils, larynx, and bronchial tubes, which are frequently associated with adenoid disease, is given in the treatment of these conditions. There is in fact no medical treatment which has more than a palliative influence. The treatment of adenoid growths is essentially surgical, and when

operative interference is necessary these cases should be referred to a specialist. The removal of these growths is not a difficult operation, but is one requiring a certain amount of experience and skill, and this is especially true since very commonly in connection with the removal of adenoids it is advisable to remove the tonsils. In very young children it is better to remove the adenoid tissue alone unless the tonsils be markedly hypertrophied and diseased. This can be done without an anesthetic.

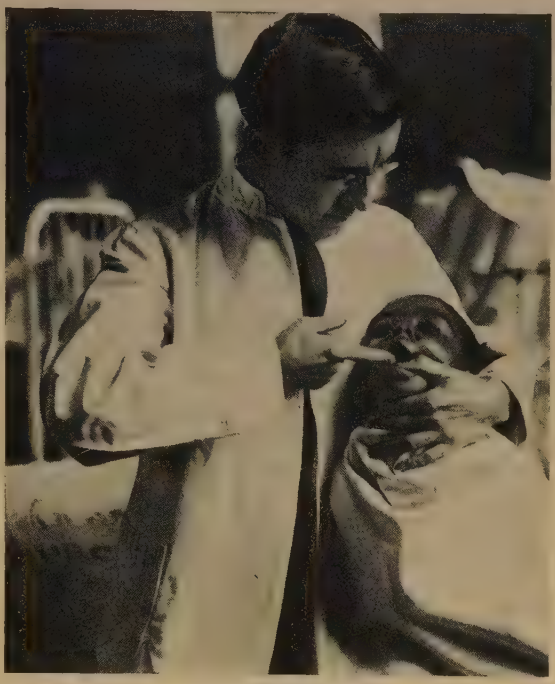


FIG. 67.—POSITION IN EXAMINATION FOR ADENOID GROWTHS.

INDICATIONS FOR SURGICAL TREATMENT.—Adenoids should be removed in all cases where either by local irritation or general constitutional disturbance they interfere with the health of the child; when the middle ear has been involved; when associated with recurring attacks of rhinitis and tonsillitis; when pronounced neurotic disorders are present, and when the nasal obstruction is such as to produce mouth breathing. In addition to this, large adenoid growths filling the vault of the nasopharynx should be removed whether or not they produce local or other symptoms.

## RETROPHARYNGEAL ABSCESS

The comparative frequency of this condition in infancy is due to the abundant distribution and marked functional activity of lymph nodes and their connecting vessels in the pharyngeal wall at this age. The diminishing frequency after the second year of life is believed to be due to the gradual disappearance and diminished functional activity of the lymphatics in the posterior and lateral walls of the pharynx during early childhood. Infection of these lymph nodes, and the consequent development of a retropharyngeal abscess, occurs more readily because of the close communication of the pharyngeal lymphatics with those of the nasopharynx. The pus-forming cocci and the influenza bacillus are the common

exciting causes. Koplik found streptococci in all of his cases. Infection is commonly secondary to ulcerative or catarrhal inflammation of some part of the throat or nasopharynx; adenoid inflammation, tonsillitis, or pharyngitis usually precede the retropharyngeal abscess. Influenza, scarlet fever, diphtheria, and the various acute infections which produce catarrhal disease of the respiratory passages may be exciting causes. Glandular tuberculosis, rickets, and other constitutional diseases, which produce malnutrition, may be predisposing factors. Age is the most notable predisposing cause. Bokay records 467 cases of which 296 occurred during the first year of life, and thereafter the disease occurred with gradually diminishing frequency, being very rare after the fifth year. These statistics, which are in accord with those of Koplik and other observers, show the comparative infrequency of this disease after the first year of life.

**Symptomatology.**—The symptoms are frequently obscured in the beginning by the causative infection. As these subside the characteristic syndrome produced by a retropharyngeal abscess begins to make its appearance. The type of temperature changes and becomes septic in character, with marked remissions and perhaps intermissions. The obstruction caused by the swelling in the pharyngeal wall causes the infant to reject its food; it lets go both the breast and the nipple to get its breath. When asleep the infant snores, and may awaken with marked difficulty in breathing, in bad cases gasping for air. Not infrequently a hoarse cough is an aggravating symptom, but it has not the barking, characteristic hoarse sound of either spasmodic or true croup. The pharyngeal stridor continues when the child is awake. The head usually is held rigidly and often inclined toward one side, and the infant cries when the neck is bent from this position. The lymph nodes at the angle of the jaw are enlarged.

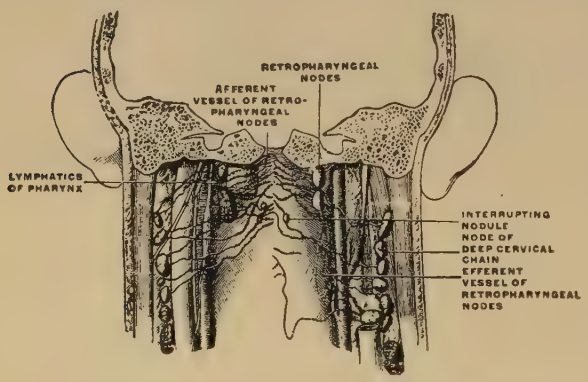



FIG. 68.—RETROPHARYNGEAL ABSCESS. (Poirier and Charpy.)

The above clinical picture can scarcely be mistaken for anything except laryngeal stenosis, and there should really be no difficulty in making the diagnosis between these two conditions. The fluctuating temperature and the absence of the characteristic croupy cough should exclude both spasmodic and true croup. Finally an examination of the pharynx of the child reveals the tense fluctuating tumor on the postero-lateral pharyngeal wall; this tumor, from one-half to one inch in diameter, may sometimes be seen by using a tongue depressor, or it may be felt by gently introducing

the finger and exploring the posterior pharyngeal wall. Great care should be exercised in making this examination not to rupture the abscess prematurely.

**Prognosis.**—When the disease is discovered early, and properly treated, there is little danger to life; nearly all such cases promptly recover. If the abscess burrows and finally ruptures spontaneously the child may be suffocated by the pus.

**Treatment.**—There should be no delay in opening the abscess after it has been discovered. In making this operation, care should be exercised to prevent the contents of the abscess from passing into the larynx. The child should be placed on its back and its head allowed to hang over the edge of the table (Rose position) and a mouth-gag introduced. With the index finger of one hand as a guide, a pointed hemostatic forceps, with the blades closed, should be forced into the abscess cavity and then the blades separated until the pus has been evacuated. The child's head should be securely held until the danger of aspirating the pus is over. General anesthesia is to be avoided (Iglauer). To prevent refilling of the abscess it is sometimes necessary to introduce a probe or finger into the opening once a day for two or three days following the operation. In rare instances the deeper lymphatics are involved and the abscess points not only into the pharynx, but also into the neck. In such cases the abscess cavity should be opened externally. This operation, however, is more difficult and should be done by a surgeon. Following the opening of such an abscess externally, it should be drained and given proper surgical treatment, until it gradually heals from within outward.

## CHAPTER XLIX

### DISEASES OF LARYNX

### ACUTE LARYNGITIS

This is an acute catarrhal inflammation of the larynx, which, in infants and young children, is commonly associated with spasm of the glottis. For this reason it is popularly spoken of as false croup, spasmodic croup, or catarrhal croup.

**Etiology.**—Laryngitis may occur as a primary infection, but, as a rule, it is secondary to catarrhal processes of the throat or nasopharynx. Rhinitis, more or less severe, is its most common antecedent. Laryngitis may also be a part of the symptom group in measles, influenza and whooping-cough. Cases associated with measles are not infrequently very violent in character. The pathogenic microorganisms capable of producing acute laryngitis are very frequently found on the normal mucous membranes of the throat and nose, and long exposure to damp, cold, or raw winds may cause a congestion of these mucous surfaces, and thus make it possible

for these microorganisms to incite a laryngitis. Laryngitis is much more common in the young child; when it does occur in older children the spasmodic element, which is partly responsible for the laryngeal stenosis, is absent, and the disease runs a comparatively mild course without alarming symptoms. The spasmodic form of this disease, called "false croup," occurs most frequently between the end of the first and the fifth year of life. It may occur even during the first year, and in neurotic children, who have been subject to croup, it may occur after the sixth year. One attack predisposes to another. There may also be a family predisposition to this disease. It is especially common in malnourished, neurotic children. The hereditary factor in many cases may be a nervous one. Rickets, chronic glandular tuberculosis, chronic anemia, and mouth breathing are important predisposing factors. Acute laryngitis not only occurs more commonly in highly nervous, malnourished children, but, in this type of child, the spasmodic element is much more severe and the attack of croup is therefore much more alarming.

The pathological condition causing this symptom group is the congested, swollen, and inflamed subglottic mucous membrane of the larynx, covered with more or less mucus. This greatly interferes with the free passage of air through the larynx and produces an inspiratory dyspnea. When to this is added spasm of the glottis, the laryngeal stenosis may for a time be almost or quite complete.

**Symptomatology.**—Catarrhal laryngitis, as it occurs in the *older child,* unassociated with spasm of the glottis, may be a primary or secondary process. It is commonly preceded by rhinitis, pharyngitis, or tonsillitis, or associated with influenza or measles. The fever, which is almost constantly present in these cases, is in no way characteristic, and adds little to the diagnosis of the disease. It is usually slight and variable, ranging from normal to 101° or 102° F.; in severe cases it may reach 104° F. High fever not infrequently depends upon the associated disease, such as influenza. The cough, dyspnea, and hoarseness are more or less characteristic symptoms. The cough is harsh and croupy in character; the most pronounced croupiness usually occurs at the onset, or at least early in the disease, and gradually disappears, leaving a laryngeal, tracheal, or bronchial cough which lasts for a week or ten days. The cough is the most persistent symptom; it may be paroxysmal, almost incessant, or nagging in character. The voice is hoarse, and, in some instances, almost lost, but the hoarseness, like the cough, is an early symptom which gradually subsides. Dyspnea is a more or less marked symptom, but is not so prominent in older children; the difficulty in breathing when it exists is associated with an inspiratory stridor. All of the above symptoms are more aggravated at night. Such is the clinical picture presented by laryngitis in the older child, and it differs little from that seen in the adult, except that perhaps in the child the cough is more croupy and associated with more dyspnea.

The clinical picture, however, produced by false croup in the young

child is very different from the one just described. There are few clinical syndromes which cause more widespread alarm in a household than an attack of spasmodic croup. The young child may go to bed without premonitory symptoms, but usually there is some slight warning during the day or late afternoon in the form of a slightly hoarse cough, which may or may not be associated with coryza or pharyngitis. The child, however, does not appear to be very ill and his temperature is but slightly above normal. He falls asleep as usual and, commonly before midnight, awakens with a harsh, croupy cough, which can be heard even in adjoining rooms. It is accompanied by great difficulty in breathing, an inspiratory stridor and very pronounced hoarseness. The difficulty in breathing increases, the face becomes cyanosed, and the child is apparently threatened with immediate suffocation. If the attack be a severe one, the mother and other attendants are thrown into a state of anxiety bordering on panic; this excites and alarms the child and perhaps aggravates the attack. The dyspnea in these cases is so pronounced that all the accessory muscles of inspiration are brought into play; there are wide flaring of the nostrils and a deep sinking in at the suprasternal notch and diaphragmatic groove. Attacks of such severity commonly last from a few minutes to one-half hour. Great difficulty in breathing, however, may continue for hours. As the attack subsides the child's breathing becomes less harsh and less labored, and it falls back more or less exhausted upon the bed and sleeps until morning. Following the attack, its clothing is wet with perspiration. In rare instances more than one attack may occur during a night. The following morning the child is found with perhaps little or no fever; it is bright, feels well and desires to get out of bed as usual. The barking cough, the dyspnea, and the hoarseness have all largely disappeared. There may be some inspiratory stridor; whistling râles may be heard in the large bronchi; hoarseness may still be present to a slight degree; the cough, which has lost its hoarse, metallic character, still persists, and throughout the day it is a prominent and irritating symptom. If the child is allowed to go about, and especially if it is not properly treated, the cough, dyspnea, and hoarseness gradually increase as bed-time approaches, and the child falls asleep and has another attack, perhaps near the same hour as the first night; it is, however, usually less severe. The second day the child again appears convalescent, but on the third night another and milder attack may occur. These croupy attacks do not usually recur for more than three nights in succession and their severity will depend upon the treatment instituted, as well as upon the physical condition of the patient; nervous, malnourished children are more subject to severe and recurring attacks. The fever may continue for three or four days and the cough usually lasts for a week or ten days. After the second or third day it gradually becomes tracheal and bronchial in character, the croupiness, hoarseness, and dyspnea being no longer present.

**Diagnosis.**—Catarrhal croup must be differentiated from laryngeal diphtheria. This, as a rule, is not difficult; there is no preliminary his-

tory of sore throat, no membrane is present on the tonsils or pharynx, and the onset of the disease is sudden. The greatest dyspnea, the hoarsest cough, and the most marked aphonia occur during the first night, and all of these symptoms almost disappear the next morning, possibly to recur the next night or two, diminishing in violence. This is in marked contrast with the gradual onset of diphtheritic laryngitis, in which the dyspnea, hoarseness, and croupiness come on slowly and gradually, from two to three days being required to produce a dangerous laryngeal stenosis; the inspiratory stridor continues during the day, although the whole symptom group may be more aggravated at night. In clearly defined cases the differential diagnosis is simple, but now and again we may have a primary laryngeal diphtheria which can scarcely be differentiated from ordinary acute laryngitis, and occasionally we may have very severe attacks of acute laryngitis, in which the symptoms, more or less modified, persist during the following day and recur with marked severity the following night. In such cases it may be absolutely impossible to make a differential diagnosis, therefore the child should be given a large dose of antitoxin (see Diphtheria), and careful bacteriological cultures from the larynx made to determine the presence or absence of the Klebs-Löffler bacillus. The diphtheria antitoxin in these cases should be given as a diagnostic measure, without awaiting the results of a bacteriological examination. A laryngoscopic examination may reveal the presence of a membrane in doubtful cases.

**Prognosis.**—The prognosis is good. Fatal cases, however, have been recorded. Bronchitis, which is the common and usual complication, prolongs the cough and other symptoms. Pneumonia, which is a comparatively rare complication, may terminate fatally.

**Treatment.**—If the child is seen for the first time during the severe spasmodic attack an emetic is indicated; teaspoonful doses of syrup of ipecac may be given every half hour until vomiting is produced; as a rule, only one dose is necessary. The emetic clears out the pharyngeal and perhaps some of the laryngeal mucus, but, more important than this, it relieves the laryngeal spasm, and thus controls the severe dyspnea in a short time. Following the emetic, the child's stomach should be allowed to rest, undisturbed by medicine or food; some hours later liquid food and the following prescription may be given:

| | |
|---|---|
| Potassium bromid | grs. 60 |
| Antipyrin | grs. 15 |
| Glycerin | ʒ i |
| Elixir of lactated pepsin | ad ℥ ii |

Sig. A teaspoonful every three hours to a child two or three years of age.

The bromid of potash and antipyrin in the above prescription are to be increased or diminished to suit the age of the child. In children over three years of age one drachm of syrup of ipecac may take the place of the glycerin in this prescription. This remedy is to be given every three

hours the first day, every four or five hours the second day, and thereafter one dose at bed-time for four or five days. This will almost always prevent second and third attacks, and will also, in the great majority of instances, prevent the first attack, if the preliminary symptoms give sufficient warning to permit the giving of several doses of this remedy during the preliminary hoarseness, which sometimes precedes these attacks. Mothers having croupy children should be provided with this mixture and advised to give it with the onset of any catarrhal condition of the throat or nose, especially if it be associated with the slightest hoarseness. In severe cases, where the emetic does not act promptly, or where it fails to give relief, a few inhalations of chloroform may be given to relieve the spasm of the glottis. This is indicated only where cyanosis is marked and difficulty of breathing, almost to the point of suffocation, is present. In some instances the laryngeal stenosis may be so great, and the diagnosis between acute laryngitis and laryngeal diphtheria so ill defined, that intubation and a good-sized dose of diphtheria antitoxin may be advisable. In ordinary laryngitis these remedies can do no harm and in diphtheritic laryngeal stenosis they are life-saving measures. If the bacteriological examination and the subsequent clinical history show the case to be one of true diphtheria of the larynx, much valuable time will have been saved, and the patient's chances for life will be much better by having given the antitoxin.

The child should be kept in bed on the day following the attack and perhaps as long as the fever and croupy cough are present. It is also almost universally recommended that children suffering from laryngitis should breathe warm, moist air. Croup tents of various kinds are used for this purpose. The bed is so tented that steam from a croup-kettle, or some other steam generator, can be directed into it. By this device the child is made to breathe warm air heavily laden with moisture. Tincture of benzoin, turpentine and guaiacol may be added to the water from which the steam is made. These drugs, when inhaled, are believed to have a soothing and antiseptic effect. While the croup tent may be of value, my own experience is that it does very little good. I have not used it in the past five years in the treatment of any kind of croup. These children do better in rooms having a temperature of about 70° F., well ventilated with fresh, comparatively warm air, carrying a moderate amount of moisture. Warm applications to the neck in the form of hot fomentations or hot poultices are of value in relieving the spasm of the initial attack as well as in preventing second and third attacks. Warm baths have a soothing and relaxing effect. During convalescence, syrup of hydriodic acid, syrup of the iodid of iron, cod-liver oil, and other tonics are of value.

The treatment of so-called croupy children during the interval between attacks is most important. They should avoid, if possible, all contagion and exposure to damp cold; should be warmly clad during the cold, damp months of winter; should live in the fresh air during the day and sleep in it during the night; they should be built up by careful feeding, regular

diet, and suitable tonics, such as cod-liver oil, malt, and iron, and, if they have a chronic tonsillar hypertrophy, or adenoid growths, these should be removed.

## EDEMA OF THE LARYNX

Edema of the larynx, incorrectly called edema of the glottis, is an edematous swelling of the submucous cellular tissues of the larynx and the aryepiglottic folds. This edema may be a simple serous infiltration, due to causes remote from the larynx, not associated with acute inflammation; in such cases the edematous mucous membrane may be pale or slightly congested. But in the most common group of cases it is secondary to an acute submucous inflammation of the larynx; in this form the exudate is seropurulent in character, and the mucous membrane of the larynx is red, swollen, ulcerated, and sometimes lacerated.

**Etiology.**—The simple serous infiltration from constitutional causes is comparatively rare in the child; it may be produced by acute and chronic nephritis, cardiac insufficiency, and by lymph nodes, and other tumors, the pressure of which prevents normal circulation in the larynx. The inflammatory form is due to infection and subsequent inflammation, or to foreign bodies in the larynx, or injuries of the mucous membrane, produced by the swallowing of corrosive chemicals or the inhalation of steam or irritating vapors. It also occurs as a rare complication of syphilis, smallpox, chickenpox, scarlet fever, diphtheria, measles, and other infections, which may excite inflammation of the laryngeal mucous membrane.

**Symptomatology.**—The most important symptom group is that produced by the laryngeal stenosis. An inspiratory dyspnea, which, in severe cases, threatens or even takes the life of the child by suffocation, is the important symptom. The child struggles for breath, is cyanotic, all the accessory muscles of inspiration are brought into play, orthopnea, laryngeal stridor, and aphonia are present. In the inflammatory cases the child complains of pain in the region of the larynx. The suddenness of the onset of the above symptom group largely depends on the exciting cause; following severe traumas it is more rapid in its development. As a rule, the diagnosis may be confirmed by introducing the finger so as to come in contact with the edematous aryepiglottic folds, or by using the laryngeal mirror or the direct laryngoscope to bring the edematous tissues into view.

**Prognosis.**—The prognosis will depend largely upon the exciting cause, and ofttimes upon the promptness with which the symptoms are relieved by surgical or other measures. Untreated cases very frequently terminate by suffocation. The milder types of the disease, due to constitutional causes, usually yield to the proper medical treatment of the exciting cause.

**Treatment.**—If marked cyanosis is present and the child is threatened with suffocation immediate intubation should be resorted to. In an instance that came under my observation about fifteen years ago a suffo-

cating edema of the larynx was produced by a small foreign body. When the tube was introduced the child was blue and seemed almost moribund, but, immediately following the introduction of the tube, the child commenced to breathe, and within one-half hour all evidences of cyanosis had disappeared. In this instance the foreign body, which was the hook of an ordinary hook and eye, was pushed into the trachea, where it remained for six weeks, producing a most irritating cough and more or less bronchitis. It was finally coughed up and the child had a rapid recovery. In some instances it may be necessary to perform tracheotomy and allow the tube to remain in the trachea until the edema of the larynx has disappeared. Scarification of the edematous tissue in some instances gives relief. All of these operative measures, however, should be carried out, if possible, by a specialist. The medical treatment of the localized inflammation in the larynx is the same as that previously recommended for ordinary laryngitis. Where heart disease and acute nephritis are the exciting causes these conditions must receive treatment. Hot baths, diaphoretics, and saline laxatives are indicated in nephritis. Digitalis, absolute rest, and a dry diet, containing not more than a pint and a half of liquid in twenty-four hours, are indicated in heart disease.

## NEOPLASMS IN THE LARYNX

This is a comparatively rare condition and is essentially a surgical one. Of laryngeal tumors, papillomata are the most common. Fibromata and malignant growths also occur.

The diagnosis in these cases is made by the slow onset of an inspiratory dyspnea, commonly associated with increasing hoarseness, and sometimes with an increasing cough. A laryngoscopic examination reveals the presence of the tumor. These cases should always be referred to the specialist for surgical treatment. The relief following the removal of papilloma of the larynx is pronounced and immediate, but frequently after some months the return of the growth is announced by the slow return of the symptoms of laryngeal stenosis, and second and third operations may be necessary. The long-continued use of Fowler's solution is said to prevent recurrence after operation.

## FOREIGN BODIES IN THE LARYNX, TRACHEA AND IN THE BRONCHI

Foreign bodies in their passage to the bronchi not infrequently lodge for a time in the larynx, producing violent irritation of this organ, sometimes resulting in edema of the larynx, but in most instances, after a violent fit of coughing and strangling with more or less dyspnea, they are either dislodged outwardly from the larynx, or pass into the trachea, where they cause more or less irritation.

All kinds of small foreign bodies may find their way into the trachea

and small bronchial tubes. Particles of food, buttons, coins, grains of corn, pebbles, and all the small objects with which children commonly play, may be aspirated into the trachea and bronchi. As previously noted, in their passage through the larynx they may excite symptoms of acute laryngeal stenosis, but after passing into the trachea and bronchi there is great variation in the symptoms they produce. As a rule, cough of a paroxysmal

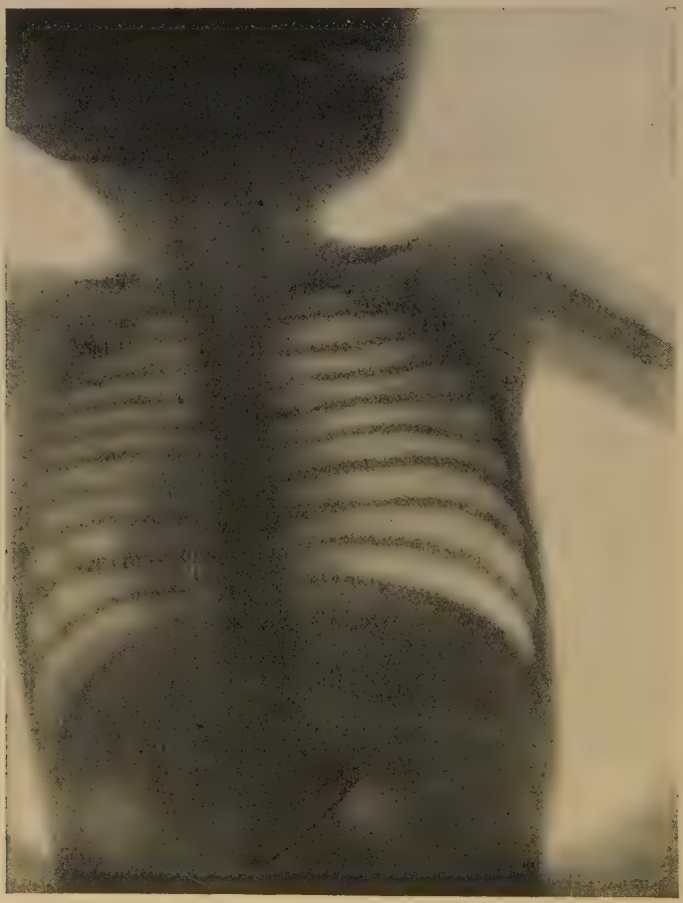


FIG. 69.—MARKED DILATATION OF THE RIGHT LUNG, PRODUCED BY FOREIGN BODY IN THE RIGHT BRONCHUS. (Iglauer.)

and aggravated type is a prominent symptom, and a whistling bronchitis of the larger tubes is nearly always present. I recently saw a case produced by the kernel of a peanut in the right bronchial tube. This child was two years of age and had been treated for some months for asthma. Its breathing was labored, and large sibilant râles could be heard over both lungs. The respiratory movements on the left side were more marked than on the right, and the vesicular murmur was markedly diminished on the right. These physical signs, in the absence of dullness, indicated plainly that

the air was passing into the left lung much more readily than into the right, and the diagnosis of a foreign body in the right bronchus was made. The X-ray picture here presented failed to reveal the presence of the foreign body, and also for a time added confusion to the clinical picture by showing that the right lung was distended and contained more air than the left. The foreign body, which was afterwards located and removed from the right bronchus, acted as a valve, which impeded expiration more than inspiration. In most of these cases, however, the reverse of this is true, and the X-ray picture may show the lung fed by the obstructed bronchus to contain less air than the other. In many instances also the foreign body is of such a character that it can be located by an X-ray picture. Increased respiratory movements on one side and diminished vesicular murmur on the other, occurring in a young child which has a troublesome cough and whistling bronchitis without fever or the physical signs of pneumonia, should be sufficient to suggest the diagnosis of an obstructed bronchus. In addition to this, there is usually a history of an acute attack of strangling with acute laryngeal irritation, as the result of "swallowing some foreign body the wrong way." In some instances these foreign bodies may remain for months or years without seriously interfering with the health of the child, and then again, after a long period of quiescence, they may produce hemoptysis, or a circumscribed bronchopneumonia. The ultimate diagnosis is made by the specialist, who locates the foreign body by the bronchoscope. In the use of this instrument it is sometimes necessary to make a preliminary tracheotomy. This is especially true of the infant, in whom it is frequently impossible or impracticable to use the bronchoscope through the larynx.

**Prognosis.**—Under proper treatment the prognosis is usually good. In the hands of a specialist, skilled in the use of the proper instruments, foreign bodies, even though they be well down in the bronchial tubes, can be rather readily removed. The prognosis in untreated cases is usually very bad. The foreign bodies in time may produce a fatal inflammation of the pulmonary tissues.

## CONGENITAL LARYNGEAL STRIDOR

This is a rare congenital condition, the etiology of which is unknown. The infantile character of the larynx persists, and the epiglottis is turned back, so that the lateral edges come in contact, leaving a very narrow opening between the aryepiglottic folds, producing a valve-like condition which obstructs the intake of air.

**Symptomatology.**—The stridor is purely inspiratory and, according to Thomson, consists of a loud crackling or croaking sound on inspiration, accompanied by the physical signs of an inspiratory dyspnea. Expiration, on the other hand, is easy and noiseless, and cyanosis, as a rule, is not marked. The stridor varies greatly in its intensity, and may at times entirely disappear and then again recur under nervous excitement, or

catarrhal conditions of the larynx. The paroxysms of dyspnea, as a rule, increase in severity during the first six months of life and then gradually subside, to disappear before the end of the second year.

This condition may be differentiated from laryngismus stridulus, thymic asthma, papilloma, and other obstructive lesions of the larynx by the fact that it begins at or immediately after birth and by the characteristic syndromes of the above-named conditions.

**Treatment.**—The child should be protected from nervous excitement and should be carefully guarded from all contagions which may produce catarrhal conditions of the nasopharynx. Fresh air, careful feeding, and all measures which will improve the physical condition of the infant will modify the severity of the paroxysms and shorten the course of the disease. The prognosis in uncomplicated cases is good.

## CHAPTER L

### BRONCHITIS

### ACUTE CATARRHAL BRONCHITIS

Bronchitis is a catarrhal inflammation of the bronchial mucous membrane which, especially in infancy, has a tendency to spread downward and involve the small bronchi.

**Etiology.**—Glandular tuberculosis, rickets, syphilis, anemia and chronic diseases of the adenoids, tonsils, pharynx, and nasal mucous membrane are the most important predisposing causes.

Infection is the essential factor in the etiology of acute bronchitis. The most common exciting microorganisms are the staphylococcus aureus, the pneumococcus, the streptococcus, the influenza bacillus, and the bacillus catarrhalis, but it may be produced by typhoid, diphtheria, and tubercle bacilli, and it is commonly associated with epidemic grippe, measles, pertussis, and scarlet fever. The influenza bacillus is commonly responsible for the chronic form of bronchitis.

The great majority of these cases occur during the winter or spring months. This is partly because the contagions which produce bronchitis are rife at this time, but also because this is the season when children are huddled together in close, ill-ventilated rooms, not only at school but in their homes. They are thereby forced to breathe an impure, germ-laden air, which is the direct cause of bronchitis. If the laity could get away from the bugbear that "catching cold" is the all-important cause of this disease and learn that the way to avoid bronchitis is to live and sleep in the open air, the morbidity and mortality from this disease would be enormously decreased. I do not wish to convey the impression that prolonged exposure to damp cold can do no harm; on the contrary, it is an important exciting cause of bronchitis. This factor, however, can only excite bron-

chitis in children who carry upon their respiratory mucous membranes one or other of the microorganisms which may cause this disease. I do not believe it is wise to expose the legs or other portions of the body to damp cold, with the idea that it exercises a hardening influence upon the child and prevents disease. It is not desirable that the skin of the child should be hardened, but only that it should breathe fresh air. During the winter months, in order that this may be accomplished with safety, it is not only wise but advisable that the child should be properly clothed so that all portions of his body may be comfortably warm.

Age is an important predisposing factor. Bronchitis is most common between the sixth month and the end of the third year of life. After this time it rapidly decreases in frequency.

**Pathology.**—The mucous membrane of the trachea and bronchi is congested and swollen, its blood vessels dilated, and its secreting structures, especially the mucous cells, increased in size and activity. The mucosa and submucosa are infiltrated with small, round cells, and with the bacteria producing the disease. The bronchi contain more or less mucus or a mucopurulent exudate. The peribronchial tissues are not involved.

**Symptomatology.**—Fever and cough announce the onset of simple bronchitis. There is nothing characteristic in the fever. It rises gradually to 102° or 104° F., is irregular in character, runs a short course, and usually reaches normal in from four to seven days. It runs an afebrile course in very young infants, and even in older ones, suffering from gastrointestinal disease, rickets, or other malnutritions. In those cases associated with influenza and other acute infections the high fever seen in the beginning is due to the general toxemia. As this subsides, the lower and irregular temperature of bronchitis may continue for a number of days before it reaches normal. If the fever remains high and prolonged, it is an evidence of a beginning bronchopneumonia, otitis media, or some other complication. The cough is the most prominent and the most troublesome symptom. It is always present, except in very young and feeble infants, and directs attention to the lungs as the site of the disease. In the beginning it is usually dry, irritating and unproductive; at this time the child is not seen to swallow following the cough. Later it is loose, less paroxysmal, and less troublesome, and usually gives more or less relief as it brings up into the pharynx some of the mucus which the child is seen to swallow following the cough paroxysm. In some instances the cough is associated with pain and more rarely with vomiting. Children under six or seven years of age do not usually expectorate; it is therefore difficult to obtain specimens of sputum for examination. If this is thought necessary, however, the sputum may be obtained by wiping out the pharynx with a gauze-wrapped finger or a cotton-wrapped probe. In this way it may be possible to decide whether the disease is produced by pneumococci, influenza bacilli, streptococci, or other microorganisms. Such information is of little or no value from a therapeutic standpoint, and this procedure therefore is hardly justifiable as a routine method of differential diagnosis.

The respiratory movements are more rapid than normal, and perhaps slightly labored. In simple bronchitis, in children over six months of age, there is practically no evidence of dyspnea; when, in such a case, therefore, the wings of the nose begin to flare, and the peripneumonic groove begins to recede with each inspiration, it is time for the physician to employ his most potent remedies to prevent the onset of bronchopneumonia. It should be remembered that there is also a form of afebrile asthmatic bronchitis, presenting all the evidences of increased labor on the part of the accessory muscles of inspiration, such as dilatation of the alæ nasi, sinking in of the suprasternal notch, and inspiratory recession of the walls of the chest in which, notwithstanding these symptoms, there is little or no danger of bronchopneumonia. This form may commonly be differentiated from ordinary bronchitis by the inspiratory stridor, the sibilant râles, and by the fact that the child has little or no fever. In very young and delicate infants there is even in simple bronchitis a slight amount of dyspnea with flaring of the nostrils and a slight recession of the peripneumonic groove.

Physical Signs.—The physical signs are well marked and by them the diagnosis of bronchitis is made. In almost every case bronchial fremitus may be felt. The vibrations of the chest wall are very significant to the experienced touch. The early sibilant and whistling râles, and the subsequent mucous râles, which may be heard in both the large and medium-size bronchi, give unmistakable evidence of this disease. Fine crepitant râles, which may occur at any time during the progress of a bronchitis, mean the onset of pneumonia. Inspection may reveal rapid breathing and a slight inspiratory retraction of the chest wall. This is especially true in young and delicate children, but when these signs are exaggerated they may be an indication of a beginning bronchopneumonia. Percussion is of comparatively little value except for determining when the disease is passing from the stage of bronchitis to that of pneumonia.

The course of simple bronchitis is usually from four to eight days; the disease, however, may be prolonged with intermissions for a period of from four to five weeks; this is common in those cases associated with subacute or chronic disease of the adenoids and tonsils. Reinfection may cause relapses in hospitals and even in private homes which are not properly ventilated and disinfected during and following an epidemic of bronchitis.

**Complications.**—Otitis media, mastoiditis, bronchopneumonia, intestinal toxemia, and gastroenteritis are common and dangerous complications.

**Prognosis.**—The prognosis is, on the whole, good, but during the early weeks of life it should be guarded, since at this time the disease may run an insidious course with little or no fever, few constitutional symptoms, and but slight cough, and yet, during all of this time, well-marked physical signs of bronchitis may be present, and a fatal bronchopneumonia may develop before the physician is aware that the infant is seriously ill.

**Prophylaxis.**—Every rhinitis, pharyngitis, or slight catarrh of the tracheal or bronchial mucous membranes should have prompt and careful treatment; this especially applies to the new-born and to syphilitic, rachitic, and other malnourished infants. Breathing pure air, living out of doors during the day, sleeping with open windows at night, and wearing, during the cold winter months, clothing that will keep the skin and body warm and dry are important prophylactic measures. All contagion should be avoided. Well infants should be kept away from persons suffering from ordinary colds, tonsillitis, grippe, and other acute infections. In children in whom the disease recurs from time to time, or who have a tendency to subacute nasopharyngeal catarrh, the throat and nose should be inspected and all diseased tissues removed.

**Treatment.**—The infant or child with acute bronchitis should, if possible, be confined to bed in a large, bright, isolated room, the windows of which are opened wide enough to let in plenty of fresh air. Care should be taken that the atmosphere of the room be not dried out with artificial heat; a moist, pure air is soothing to the irritated bronchial mucous membranes. As a rule, all that is necessary is to admit the outside air; this generally secures sufficient moisture. Where this cannot be satisfactorily accomplished, the air of the room may be moistened by heating water in an open vessel. The infant or child should be clothed so that its body will be kept warm whatever may be the temperature of the room. In winter the bedroom should be kept between 60° and 70° F.

The medical treatment is largely symptomatic. In the beginning, if the child be suffering from some acute intoxication, such as influenza, which produces high fever and marked discomfort, phenacetin may be given for one or two days but should not be continued longer. A safe prescription in these cases is guaiacol carbonate, 1 grain; salol, 1 grain, and sugar, 1 grain. This dose may be given every three or four hours to an infant under one year of age, and may be increased to suit the age of the child. The cough may be allayed by the use of bromid of potash, 45 grains; tincture of belladonna, 15 minims; glycerin, 2 drachms, and elixir of lactated pepsin, enough to make 2 ounces. A teaspoonful of this mixture may be given every three hours to an infant one year of age. For older children, syrup of ipecac, 1 drachm to the ounce, may be added to this prescription, and the doses of the other ingredients increased to suit the age of the child. The opium preparations are almost never indicated in children under two years of age, but for sturdy children, over this age, 1 to 2 drachms of camphorated tincture of opium may be added to the above prescription. In the chapter on Bronchopneumonia I have spoken most decidedly concerning the danger of giving opium, cough syrups, ammonium carbonate, ammonium muriate, tartar emetic, squills, and ipecac, and what is said there applies with almost equal force to their use in ordinary bronchitis. These remedies are rarely indicated, and I feel quite sure that more harm than good is done by their indiscriminate use in children under two years of age.

In beginning the treatment, the gastrointestinal canal is to be thoroughly unloaded by a dose of castor oil, and throughout the disease this dose is to be repeated every three or four days to prevent intestinal infection by the mucus and pus which have been coughed up and swallowed. Warm tub baths or warm sponge baths are very grateful, and serve a useful purpose in the treatment of bronchitis. They quiet the nervous system, promote the action of the skin, and act as a general tonic. Cold baths and cold packs are not indicated; this is especially true in infants under eighteen months of age. Antipyretics are not needed to reduce the temperature. Inunctions of guaiacol, 1 drachm to the ounce of anhydrous lanolin, should, in one-half-teaspoonful doses, be thoroughly rubbed into the skin of the chest, night and morning. A light oilskin jacket, lined with a thin layer of cotton-wool, is of value, and is especially indicated in the infant and young child during the cold winter months when the fresh-air treatment is being given. If at any time the symptoms indicate that a broncho-pneumonia may be developing, flaxseed poultices are to be used as directed in the chapter on Bronchopneumonia.

### CHRONIC BRONCHITIS

Chronic bronchitis is comparatively rare in children. The only form that here need be mentioned is the asthmatic bronchitis previously referred to. Its treatment may require carbonate of creosote, syrup of hydriodic acid, cod-liver oil, malt, and other tonics. A warm, equable climate is of value.

### MEMBRANOUS BRONCHITIS

In this condition the mucous membrane of the trachea and bronchial tubes is covered with a fibrinous deposit. It is comparatively rarely seen, except in diphtheria, where the diphtheritic membrane may extend down into the bronchial tree. It also occurs occasionally in croupous pneumonia and some of the other acute infections. The symptoms are those of a severe bronchitis, and the diagnosis is made on the expulsion of fibrinous casts.

The treatment is that of severe bronchitis in addition to the specific disease which produces it.

## CHAPTER LI

### LOBAR PNEUMONIA

*(Croupous Pneumonia, Fibrinous Pneumonia)*

**Etiology.**—This is an inflammation of the lungs which, in from 90 to 95 per cent. of the cases, is caused by the Fränkel diplococcus pneumoniæ (pneumococcus). It is believed that in the majority of these cases

there is a general infection with the pneumococcus, and that the pulmonary lesion is but a local expression of a general constitutional disease. From this viewpoint we may class the disease among the acute infections. The lungs are the favorite site for the local lesion, because the pneumococcus commonly finds its entrance through these organs, and also perhaps because they are especially susceptible to pneumococcic inflammations. Clinical experience, as well as pathological and bacteriological research, has taught us that it is better to consider this disease from the broad viewpoint of a general infection, and as such we shall speak of it in this chapter. It should be remembered, however, that the same croupous or fibrinous lesions found in lobar pneumonia may perhaps, in a very small percentage of cases, be produced by organisms (Friedlander's bacillus, Pfeiffer's bacillus, and streptococci) other than pneumococci; at least it may be said that pneumococci have not been found in these cases, and again, on the other hand, it should be noted that the pneumococcus, in its onslaught upon the lungs, is associated in its destructive processes with streptococci, staphylococci, the Friedlander bacillus, and other organisms which so commonly produce secondary infections.

Exposure to damp cold, the inhalation of irritating particles, and all causes that produce congestion or catarrhal inflammation of the respiratory passages may be important predisposing causes in that these conditions incapacitate the mucous membranes for resisting the pneumococcus. Influenza, measles, and other acute infections may act in the same way. Croupous pneumonia is comparatively rare during the first six months of life, but is very common from that time to the end of the second year, and is comparatively frequent up to the fifth year. It is more common during the winter and spring, and occurs with equal frequency in robust and feeble children.

**Pathology.**—The most important pathological condition is the pneumococcic septicemia. The pneumococcus may be demonstrated in the blood in most of these cases, and it is found in the pulmonary lesions in nearly all cases that come to post-mortem examination. In fatal cases it is commonly associated with streptococci, staphylococci, or other organisms. The pulmonary lesions do not occur so early and are not so frank and apparent as in the adult. But the pulmonary lesion, when it does occur, is similar to that of the adult and therefore requires no detailed description here. The pulmonary inflammation in lobar pneumonia begins in the lung tissue and not in the small bronchi as in bronchopneumonia. It spreads more or less rapidly through the lung by continuity of surface, usually confining itself to one lobe. A fibrinous exudate is thrown out, which, with the other inflammatory products, results in an airless or consolidated condition of the part of the lung affected. The inflammatory lesion passes through the stages of congestion, red and gray hepatization and resolution. The pleura in nearly every case is more or less involved, usually so slightly, however, as not to affect the course or prognosis of the disease. The pleurisy may be fibrinous, serous or purulent

(empyema). When empyema occurs this complication becomes the dominant symptom group, greatly exceeding in importance and danger the original condition.

The whole or part of one lobe may be involved, or, less commonly, more than one lobe in the same or in different lungs may be affected. Very rarely an entire lung may be consolidated. The central area of a lobe may be involved for some time before the disease reaches the surface and produces physical signs. It is probable, however, that most of these so-called "central" pneumonias are cases of general pneumococcic infection in which the pulmonary lesion appears as a late manifestation. The pulmonary lesion occurs with about equal frequency in the right upper and left lower lobes; in about two-thirds of the cases the disease begins in one or the other of these sites. The left upper and right lower lobes are affected with about equal frequency, the disease occurring in these locations about half as frequently as it does in the right upper and left lower lobes. Much less commonly, the right middle lobe is the first part of the lung to be affected. The statistics of different writers vary somewhat with reference to the frequency of the involvement of the right upper and the left lower lobes; in infants the right upper lobe is more frequently involved, in children the left lower lobe is the favorite site.

**Symptomatology.**—General Symptoms.—Within a few hours the child presents the appearance of being acutely ill; it is feverish, dull, listless, and gives little heed to its surroundings. In older children a distinct rigor, followed by a sudden elevation of temperature and pain in the side, may call attention to the lungs as the site of the disease, and soon these symptoms may be followed by cough, and later by rusty sputum and the physical signs of acute lobar pneumonia. We are here interested, however, more especially with the symptoms of this disease as it appears in the infant and young child, in whom the early symptom-complex is very different. The chill is rarely present; a convulsion may occasionally take its place, or, with the sudden rise in temperature, the child may feel chilly, have cold extremities, and its face show a pinched expression. With the sudden onset of acute symptoms, the evidences of a severe acute intoxication are well marked; there is high fever and the infant is dull, stupid, more or less prostrate, and is little, if at all, interested in its food or toys; anorexia, to the extent of absolutely refusing food, is common, but thirst is increased. Cough and pain in the side are not usually present during the first days of the disease. Vomiting is an early and common symptom but does not, as a rule, persist after the second day. Diarrhea is rather common in infancy, but constipation is more frequent in older children. Associated with the high fever, which may reach 104° or 105° F. in the first twenty-four hours, there is a rapid increase of frequency in both the respiration and pulse, but marked dyspnea, with flaring of the nostrils and retraction of the lower part of the chest, is not, as it is in bronchopneumonia, an early and prominent symptom. The normal ratio between the pulse and respiration is disturbed; this is a very valuable early

symptom. The respiration commonly ranges from 50 to 70, and the pulse from 140 to 160; the respiration-pulse ratio is thereby increased from the normal 1 to 4, to 1 to 2½ or 3; later in the disease this ratio may be as 1 to 2. On the second or third day there is more or less cough, and the characteristic expiratory grunt may be associated with slight dyspnea, which tends to confirm the diagnosis and stimulate the physician to a careful search of the chest for the earliest physical signs associated with the pulmonary lesion. The fever continues high for from five to eight days, and is perhaps during this time more sustained than in any other disease of early childhood. As the physical signs in many cases appear late, the diagnosis must be made or at least suspected by the general

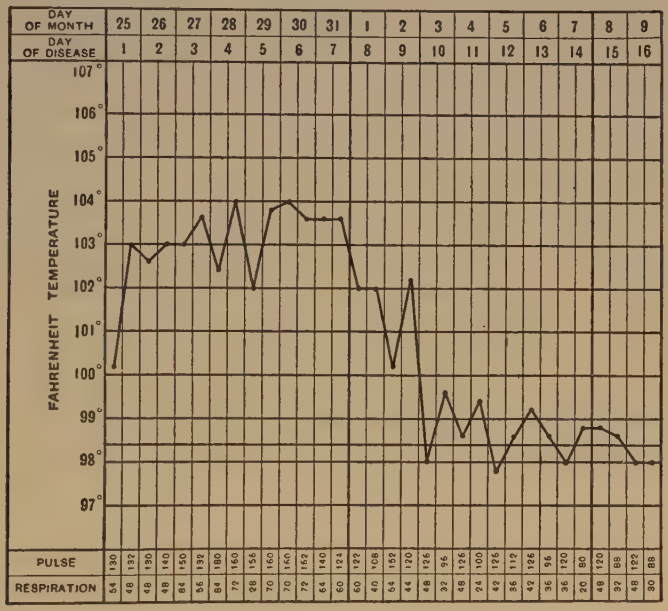



Fig. 70.—Lobar Pneumonia; Child Two Years of Age.

symptom-complex as above outlined. The sudden onset, with high and sustained fever, the marked constitutional depression with listlessness and sometimes stupor, the disturbance in the pulse-respiration ratio, the cough, the slight dyspnea, and the expiratory grunt are usually sufficient to make a tentative diagnosis of pneumonia.

*Fever.*—In the great majority of cases the temperature rises suddenly so that within the first twenty-four or thirty-six hours it may reach 105° or 106° F. It continues high, fluctuating slightly, until the crisis occurs on or about the seventh day. At this time it is not unusual for the temperature to fall within twelve or twenty-four hours from 105° F. to below normal, and with this fall there is a rapid decrease in the pulse and respiration. This subnormal temperature may continue for a day or two,

or it may be varied by slight elevations before it becomes normal. An uninterrupted recovery commonly follows. In not every case, however, is this typical temperature curve observed, irregularities of various kinds being possible. In a minority of cases the fever falls by lysis with sharp variations in the temperature until it finally reaches normal. Abortive cases occur in which the fever may continue high for two or three days, and then suddenly fall to normal. In other instances the fever may be prolonged, with irregularities, or even short intervening normal periods, for two or three days; these cases are sometimes spoken of as relapsing pneumonias. They represent those cases in which there is an extension of the pneumonic process to other parts of the lung after the focus of primary pulmonary inflammation has almost or quite run its course.

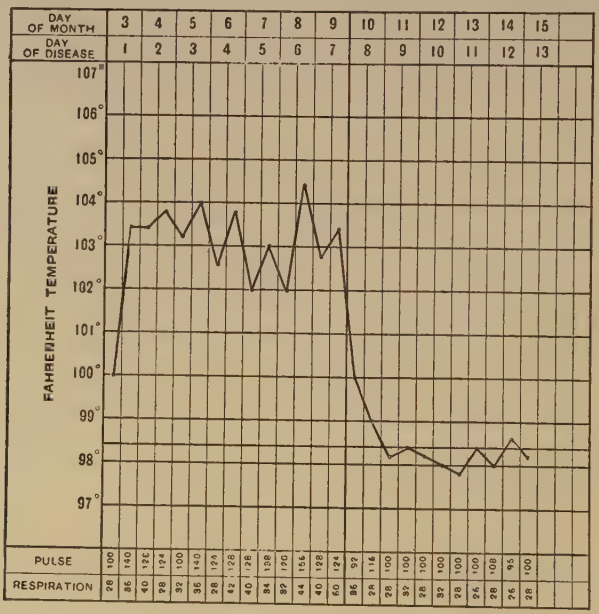



Fig. 71.—Lobar Pneumonia; Child Four Years of Age.

*Respiration.*—This, as previously noted, is accelerated out of proportion to the increase in the pulse rate. It commonly varies between 40 and 80; the younger the infant the more rapid the respiration. The tachypnea or rapid breathing, however, in this disease is associated with comparatively little dyspnea, and in this it differs markedly from bronchopneumonia. In some cases, however, as the disease progresses, the accessory muscles of respiration are brought into play and flaring of the nostrils is noted, but the marked drawing in of the chest at the diaphragmatic groove, which is so characteristic of bronchopneumonia, is slight or not at all present. The pause, however, which occurs at the end of inspiration, followed by shallow expiration, and associated with an expiratory grunt, is very characteristic of lobar pneumonia.

*Cough.*—During the first day or two cough may be entirely absent, but sooner or later it becomes a noticeable symptom, not infrequently associated with pain in the side or abdomen. The cough is one of the last symptoms to disappear and is not infrequently more pronounced during convalescence than during the height of the fever. Sputum is difficult to obtain, as young children do not expectorate. Occasionally, however, specimens may be secured when the child vomits, or cough may be excited by introducing a gauze-wrapped finger into the pharynx, and wiping up the sputum as it is brought up in this way. Sputum thus obtained may present the typical rusty appearance, and pneumococci in great numbers may be demonstrated in it.

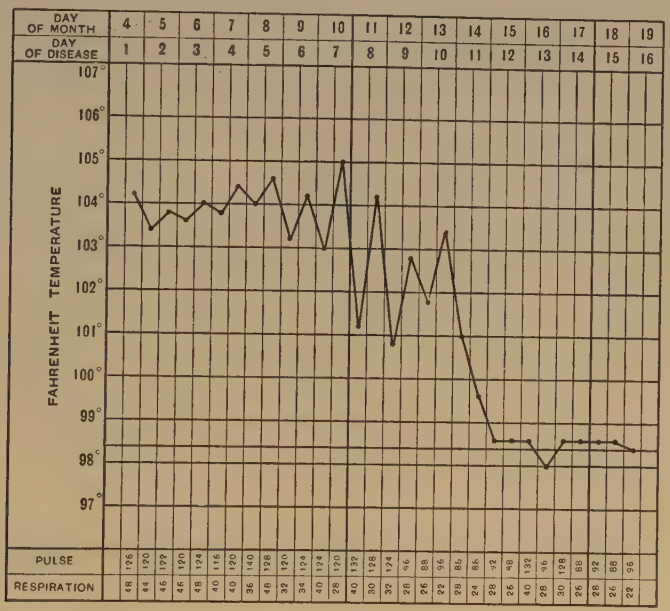



FIG. 72.—LOBAR PNEUMONIA; CHILD TEN YEARS OF AGE.

THE BLOOD.—Blood cultures will, in the great majority of cases, demonstrate the presence of the pneumococcus, but this procedure is not necessary or justifiable for routine diagnosis. A well-marked polynuclear leukocytosis is present, which may reach from 40,000 to 50,000 to the cubic m. m. A high leukocyte count is a favorable indication, while a low leukocyte count, with well-marked physical signs of lobar pneumonia, is an unfavorable sign.

URINE.—The urine, in many of these cases, contains a trace of albumin with occasional hyalin and granular casts. This febrile or toxic albuminuria is of comparatively little significance as it disappears shortly after the crisis. Acute nephritis may occur as a rare complication.

PHYSICAL SIGNS.—In examining the chest of infants and young children the physician must keep in mind the fact that the inspiratory sounds

at this period of life are loud and coarse in quality, and that this is especially noticeable on the right side, beneath the clavicle and over the spine of the scapula. This normal puerile breathing may easily be mistaken for bronchial or tubular breathing, such as is found in pneumonia. The physical signs of pneumonia in infancy are similar to those occurring in the adult, but they not infrequently appear so late that they are of little value in the early diagnosis of this disease, and, in some instances, they may never be discovered or they may be so evanescent as to be misleading. These facts, however, only make it more imperative that the physician should carefully search the chest, day after day, in all suspected cases.

*Percussion.*—It is important to remember that light percussion may disclose small areas of dullness where strong percussion fails. In the early diagnosis of croupous pneumonia, however, percussion is little to be relied upon, but as consolidated areas appear they may be discovered, and in advanced cases the dullness may be well marked over one or more lobes of the lungs.

*Auscultation.*—Auscultation is of great value. By it one may sooner or later discover crepitant and subcrepitant râles, the most valuable of all physical signs. These are commonly heard at the base or apex of the lungs; sometimes they are first heard in the axillary region; the whole lung should, day after day, be carefully searched for them. Bronchophony is an early and valuable physical sign, and bronchial breathing may later be made out over the consolidated lung. Coughing, and crying, by producing deep inspirations, may serve to bring out or make the auscultatory signs more pronounced.

**Complications.**—Pleurisy is the most common complication. In most instances it adds to the discomfort rather than to the seriousness of the disease. In a small minority of the cases, however, instead of a simple fibrinous or mild serous pleurisy, we have a complicating empyema, which at once becomes the most serious feature of the disease. In every instance, where delayed convalescence is associated with an intermittent fever, empyema should be suspected and carefully searched for. Otitis media is a common complication. Meningitis is a rare complication, but meningism is not uncommon. Arthritis, peritonitis, pericarditis, endocarditis, osteomyelitis, abscess of the liver, and diseases of the accessory sinuses of the nose are possible complications, all of which may be excited by the pneumococcus. Bronchopneumonia is a common and very dangerous complication in children under two years of age, but it is more often seen in hospital than in private practice. Pneumonia occurring in tuberculous children may be prolonged and may develop into an active, advancing pulmonary tuberculosis.

Splanchnic, or vasomotor, paralysis, as Romberg and Pässler have shown, may result from the action of pneumococcus toxins on the vasomotor center. In this condition the blood is withdrawn from the heart and the general circulation into the dilated veins of the splanchnic area,

and the heart, because of the scarcity of blood, becomes feeble and irregular in action, and death may result. This condition occurs only when there is a profound general pneumococcus infection.

**Differential Diagnosis.**—In some cases vomiting, slight intestinal disturbance, fever, and general toxemia may suggest a gastrointestinal intoxication, but since a laxative with abstinence from food makes little or no impression upon the fever and constitutional symptoms, intestinal toxemia may thereby be quickly eliminated. In some cases the vomiting, the stupor, the delirium, the rigidity of the muscles of the neck and upper part of the back, to which may be added a certain degree of opisthotonos, very strongly suggest meningitis. These symptoms, however, come on slowly, so that by the time the meningeal syndrome is closely simulated one can, as a rule, find the physical signs of pneumonia in the lungs. In other cases the vomiting, abdominal pain and distention, tenderness, and slight resistance on the right side over the head of the colon may strongly suggest appendicitis; many such cases have been operated upon. The well-known tendency of the child to refer thoracic pain to the abdomen makes the danger of mistaking a pneumonia for an appendicitis more probable. Griffith says: "The distinction is to be made by giving due consideration to (1) the sudden rise of temperature to 103° F. or thereabouts, and the tendency to maintain this degree; (2) the acceleration of respiration, which is out of proportion to the pulse rate or pyrexia; (3) the relaxation of the abdominal walls between the respirations; (4) the diminution or the disappearance of tenderness on deep pressure with the flat of the hand; (5) the possible presence of cough. Finally no operation for appendicitis should ever be performed until after a careful or perhaps repeated examination of the lungs has been made." For the further differential diagnosis see Bronchopneumonia.

**Prognosis.**—The prognosis in uncomplicated lobar pneumonia in the infant and young child is vastly better than it is in the adult. The great majority of these cases get well, and there is perhaps no serious disease of infancy and childhood in which the convalescence is so rapid and so satisfactory. Following the crisis the child improves so rapidly that it is difficult to keep him in bed for the seven to ten days necessary to insure satisfactory convalescence. The younger the infant the more unfavorable the prognosis, so that in infants under one year of age, especially where there is well-marked consolidation of the lung with high fever and rapid respirations, it is well to give a guarded prognosis, since a considerable percentage of these cases end fatally. In general terms it may be said that in infancy the death rate is from 10 to 20 per cent., and in children from 3 to 6 per cent.; the lower figures in each case refer to children treated under favorable hygienic conditions in their own homes.

**Treatment.**—It should be remembered that lobar pneumonia is a self-limited disease for which we have no specific and in which we may do great harm by over-medication. The treatment must be largely hygienic and

sustaining, and even the symptomatic treatment which is indicated at every stage must not be overdone.

Hygienic Treatment.—The hygiene of the sick room is all-important. The child should be put to bed in a large, bright room, under the care of a trained nurse, instructed to keep the room quiet and free from unnecessary visitors. The most important agent is fresh air. This fact was long ago emphasized by Northrup, who has continued for the past ten years to be the most ardent advocate of the fresh air treatment not only of pneumonia, but of many other acute diseases; as he says, the air in the room "should be fresh, cool, and flowing." In other words, the patient is to be in a room with windows open night and day, even though the temperature in the room, during the winter months, falls as low as 60°F.; with proper indoor heating apparatus the room may be kept at a temperature of 60° to 65°F., with fresh, cool, "flowing air" coming in through two or more windows. In carrying out this treatment it is evident that not only the garments, but the bed-clothing, of the child should be adapted to the temperature of the room. The body of the child must be kept comfortably warm, while it is breathing the fresh, cool air. If necessary, hot water bottles and warm blankets should be used on very cold days. The nurse on duty in such a room should dress to protect herself from the cold. When this treatment was coming into vogue, a number of years ago, the physician found it difficult to overcome the prejudice against "catching cold," but the success of the outdoor or fresh air treatment of this disease has been so clearly demonstrated in the last ten years that the laity have now come to accept it without protest.

Dietetic Treatment.—The importance of the dietetic treatment is emphasized by the fact that gastric and intestinal disturbances are common, and that anorexia is often so pronounced, that the child refuses all food; in the face of these conditions the physician is called upon to combat, with proper food and nourishment, an exhausting disease, which may run for a week or more with high fever, and severe toxic symptoms. Infants and young children can usually be induced to take a moderate amount of liquid foods, such as modified milk, beef juice, meat broths and egg albumin; it is important, however, that these foods should not be given at such short intervals, and in such quantities, that the weakened digestive capacity of the infant will be overtaxed, and intestinal fermentation with gaseous distention be thereby produced. In underfed infants whiskey or brandy is indicated from the beginning of the disease. In my experience, good rye whiskey acts better than brandy; when mixed with water and a little sugar it is readily taken, and produces no gastrointestinal disturbance; where the carbohydrate intake is small whiskey acts as a food rather than as a stimulant, and thereby furnishes energy to the cells and prevents emaciation and loss of strength. It is my custom to give to infants, between one and two years of age, 20 or 30 drops three or four times in the twenty-four hours; this dose may be increased with increasing toxemia and prostration.

Cool water to drink and plenty of it was long ago recommended in the treatment of pneumonia. While these children have little or no appetite, they are thirsty, and will drink a large quantity of cool water, the intake of which helps to reduce the fever and to diminish the toxemia, as it promotes the excretion of toxins through the skin and kidneys.

Local Applications.—Poultices and oil silk jackets encasing the chest are not indicated in this form of pneumonia. Hot water bottles, electric pads, small poultices and mustard plasters may be used for the relief of the pleuritic pain, when it is severe enough to interfere with sleep. Mustard plasters, which are so universally recommended as counter-irritants, should be strong enough to produce only redness of the skin without blistering; two parts of flour and one part of mustard made into a paste and spread between two layers of gauze may be used for this purpose. I have rarely found it necessary to use counter-irritation in the treatment of lobar pneumonia in children. Priessnitz's applications are of great value when the fever is high and the respirations rapid. They are applied by dipping a piece of light flannel in water (temperature about 70°F.), wringing it out, applying it to the entire chest of the child, and covering it with a dry flannel. The wet flannel may be changed from every half hour to three hours, as the symptoms demand.

Antipyretics.—It is most important that the physician should not center his attention upon the high temperature and attempt to beat it down with cold water and other antipyretics. A temperature of 103½° or 104°F. in lobar pneumonia, as a rule, does not require antipyretics; hyperpyrexias of 105° and 106°F., however, demand treatment. The use of baths for the reduction of temperature will depend altogether upon the manner in which the individual case responds to this treatment. If the high temperature is associated with marked nervous symptoms and other evidences of profound toxemia, hydrotherapy will, in the great majority of cases, give great relief, not only by reducing the temperature, but also by quieting the nervous symptoms and stimulating nutritional processes. If such a favorable result follows the use of the bath, it may be used as indicated throughout the course of the disease. Many cases suffering from nervous symptoms and severe toxemia are benefited by a hot tub bath twice in twenty-four hours, but when these symptoms are associated with high temperatures sponging with alcohol and water, or what is much more effective, the cold pack, may be resorted to three or four times in twenty-four hours. In giving the cold pack, the body of the child should be wrapped in a bath towel wrung out of cold water, and over this a light dry blanket should be wrapped. The towel may be removed after one-half hour and the child sponged off with alcohol and warm water. Ice-caps may be applied to the head with benefit, especially in those cases with high temperature and pronounced nervous symptoms.

The coal-tar antipyretics are almost universally condemned by writers upon this subject, and yet they are almost universally used by the general practitioner, and I am inclined to believe, from my own experience, that

in certain cases of high temperature with pronounced nervous symptoms phenacetin may be used to advantage, especially in older children. There is no question as to the sedative and antipyretic action of this drug. It will ofttimes produce a quiet sleep by relieving headache and other pain, and I am of the belief that the sleep thus produced does more good than the depressing effect of the phenacetin can do harm. Under two years of age this drug should not be used, but in older children it is to be recommended, not as an antipyretic, but occasionally to relieve the pain, restlessness and nervousness, which prevent sleep.

Medical Treatment.—With the onset a cathartic should be given. Castor oil is to be preferred, if nausea and vomiting do not prohibit its use; if castor oil is contraindicated, calomel should be given, followed by a dose of Rochelle salts. Throughout the course of the disease the gastrointestinal tract should be carefully watched and cathartic medication resorted to, to prevent abdominal distention, to overcome constipation, and to clear the canal of fermenting food stuffs, which may be adding an intestinal toxemia to the existing disease; this is especially important in infants and young children. Care should be exercised that this laxative treatment be resorted to only when necessary, as harm may result from unnecessary catharsis.

Quinin is a remedy of value in the routine treatment of pneumonia in children over two years of age. It should be given in the form of euquinin to young children, and in the form of the sulphate or bisulphate to older ones. The disagreeable taste of this drug is a rather serious objection to its administration in a disease in which so much depends upon the giving of proper foods and stimulants, and its greatest value, therefore, is in children who are old enough to take it in capsule form. The vaccine treatment is contraindicated in acute forms of pneumonia. In the chronic forms, however, which are occasionally seen in older children, an autogenous vaccine, or the pneumococcus stock vaccine, may be used at times with great advantage as directed under vaccine therapy. The use of antitoxic serums has not been followed by appreciably good results. Carbonate of guaiacol or creosote may be given internally, but their value when administered in this way is so problematical that it is much better to administer guaiacol by inunction. One drachm of liquid guaiacol when thoroughly incorporated with 1 ounce of anhydrous lanolin may, as a routine measure, be administered night and morning, by inunction in ½- or 1-drachm doses, as recommended in the chapter on Therapeutics of Infancy and Childhood.

Tincture of strophanthus is, in my opinion, the most valuable stimulant we have in this disease. I have used it as a matter of routine treatment in every case of pneumonia which I have seen in the last fifteen years. I do not believe that it is contraindicated in the beginning, or that it should be given only when cardiac failure commences. To infants between one and two years of age one drop should be given every four to six hours; between three and four years, two drops. As the disease ad-

vances, this dose may be doubled in frequency rather than in size, so that a child of two years, as it approaches the crisis, will be taking two drops every two or three hours. In severe cases, where the toxemia is great and the respirations rapid, sulphate of strychnin is a valuable respiratory stimulant and general tonic; for a child two years of age 1/150 of a grain may be given with whiskey at three- or four-hour intervals, or 1/300 of a grain may be given hypodermically at six-hour intervals. Caffein-sodium-benzoate or salicylate (in 1-grain doses by mouth, or ½-grain doses hypodermically, for a child four years of age) is one of the most valuable circulatory stimulants. It is indicated in severe general pneumococcic toxemia, in which there is danger of vasomotor paralysis. It should always be used if the pulse becomes feeble and intermittent.

Oxygen is a valuable respiratory stimulant, but the indications for its use have been greatly diminished by the fresh-air treatment of this disease; it may, however, be used in tiding desperate cases over the crisis; it is indicated when the respirations are very rapid and cyanosis is marked. Nitroglycerin is also recommended in threatened collapse; 1/300 of a grain may be given hypodermically to a child three years of age. As previously noted, whiskey in large doses, 1 or 2 drachms, may be used as a stimulant and to counteract the toxemia in severe cases.

Sedatives other than the bromids and belladonna have no place in the treatment of pneumonia in infancy. For children between the age of one and two years, three or four grains of bromid of potash and one minim of tincture of belladonna may be given at three- or four-hour intervals, to allay the cough and nervousness. These drugs should be given in some palatable vehicle, such as the elixir of lactated pepsin, so as not to irritate the child or disturb the stomach. Opiates are, in my opinion, dangerous drugs in the treatment of the pneumonias of infancy. They are rarely, if ever, indicated under two years of age. In older children, between three and five, codein, 1/10 of a grain, paregoric, fifteen or twenty drops, or some of the other preparations of opium may occasionally be indicated to relieve the pain caused by the cough or by a complicating pleurisy.

Expectorants, such as ammonia, ipecac and squill preparations, are contraindicated, since by disturbing the gastrointestinal tract they do more harm than good.

Treatment of Convalescence.—The only treatment commonly necessary during convalescence is to keep the child in bed for a week and let him have plenty of fresh air and a carefully selected nutritious diet within the range of his digestive capacity. If, however, the child does not rapidly regain its strength, is anemic, and has little appetite, it may be benefited by such tonics as malt and iron, malt and cod-liver oil, syrup of the iodid of iron, or syrup of hydriodic acid. In weak children and those predisposed to tuberculosis, creosote or the benzoate or carbonate of guaiacol may be given with advantage.

# CHAPTER LII

## BRONCHOPNEUMONIA

Bronchopneumonia, next to gastroenteritis, is the most common of the serious disorders of infancy. It is a disseminated and lobular inflammation of the lungs, which usually follows and is always associated with a bronchitis of the smaller bronchi. It is, in the vast majority of cases, a direct sequel of some form of bronchitis. The many causes of bronchitis are, therefore, its more or less direct etiological factors. It is a syndrome rather than a distinct disease, in which the pathological processes are excited and kept up by a variety of microorganisms. The pneumococcus, which, as has been previously noted, is almost the sole cause of lobar pneumonia, is the exciting cause of bronchopneumonia in a considerable number of cases. The so-called primary cases of bronchopneumonia occurring in infants, are almost all due to this organism, and should properly be classified with the lobar pneumonias under the term pneumococcic infection. There are many difficulties, however, in adopting such a classification in a text-book. The various forms of pneumonia have for generations been classified according to their anatomical findings, and, while the terms lobar and bronchopneumonia may be confusing, and actually misleading, in the light of the present-day conception of these diseases, they are sanctioned by long usage and cannot, in the present state of our knowledge, be replaced by a practical etiological (bacteriological) classification, although the present trend of bacteriological research indicates that such a classification may be adopted in the future. It is better, therefore, from the standpoint of the clinician, to continue to use the terms lobar and bronchopneumonia. It is to be understood that lobar pneumonia is used in the broad sense previously described, and that the term bronchopneumonia is used to mean an inflammatory process of the lobules and of the smaller bronchi, from whatsoever cause this inflammation may be produced. This gives the clinician an opportunity to still further classify his bronchopneumonias, not only with reference to the age and physical condition of the child, but also with reference to certain more or less definite etiological factors which produce variations in the clinical types of this disease.

**Etiology.**—Age is an important predisposing factor. Bronchopneumonia is comparatively rare during the first few months of life; it is most prevalent during the second six months, and continues to be very common during the second year; thereafter it occurs with rapidly decreasing frequency up to the sixth year. About three-fourths of the cases occur during the winter and spring months. This is due to the fact that the acute infectious diseases and bronchitis are more prevalent at this time. Bad hygienic surroundings are very potent etiological factors. This disease is found very much more frequently in hospitals, institutions for children, and tenement houses than it is in the homes of the well-to-do. Gastro-

intestinal disorders, glandular tuberculosis and all forms of malnutrition, especially rickets, may predispose to bronchitis, and at the same time diminish the natural powers of resistance of the infant, so that a bronchitis may readily develop into a pneumonia. Bronchopneumonia usually follows a simple bronchitis or a bronchitis produced by measles, influenza, pertussis, diphtheria, scarlet fever, or some other acute infection. The bronchopneumonia of measles occurs, as a rule, during the stage of eruption, but is not infrequently overlooked until the eruption has subsided. Bronchopneumonia occurs as a complication of whooping-cough during the height of that disease, when the paroxysms are severe and the resistance of the child is somewhat reduced by the long siege of coughing. It is more likely to occur in young and delicate infants, especially in those who are suffering from a glandular tuberculosis; it is more dangerous than the bronchopneumonia following measles. Influenzal bronchopneumonia may be due to the influenza bacillus, unassisted by other microorganisms; as the disease progresses, however, secondary infection usually occurs. True influenzal bronchopneumonia is comparatively rare, but may occur during an epidemic of this disease; it is more prevalent in older children than in infants. The bronchopneumonia following diphtheria and scarlet fever is usually severe; it is usually a fatal complication when it occurs in cases of laryngeal diphtheria, following intubation or tracheotomy. The most important fact to keep in mind concerning the secondary bronchopneumonias caused by the acute infections is that they are nearly always mixed infections, and that in the vast majority of cases, especially in measles, pertussis, and influenza, these bronchopneumonias may be prevented by fresh air and proper hygienic surroundings, and may be produced by confining patients suffering from these acute infections in close, ill-ventilated rooms, and especially by associating them with other children suffering from bronchopneumonia, or any form of infection in which the septic cocci are causative factors. The microorganisms most commonly associated with the destructive processes in bronchopneumonia are streptococci, staphylococci, pneumococci, Friedlander's bacilli, influenza, typhoid, and diphtheria bacilli.

**Pathology.**—The important difference between bronchopneumonia and lobar pneumonia consists in the primary involvement of the finer bronchi in the former disease, while in the latter the inflammatory process spreads more or less rapidly through the lung tissue without the intervention of a catarrhal inflammation of the finer bronchi. From this it would appear that in bronchopneumonia the infectious agent reaches the lung tissue through the small bronchi, while in lobar pneumonia it probably reaches the lungs through the lymph or blood channels, causing primary inflammation of lung tissue, which spreads rapidly and by extension includes the fine bronchi in the inflammation. This essential difference in the pathological anatomy of the two diseases would indicate that lobar pneumonia is really a primary acute pneumococcic infection which, as a rule, finds more or less extensive local expression in an acute inflammation and re-

sulting consolidation of lung tissue, and which involves and is usually confined to either the whole or part of one lobe; on the other hand, bronchopneumonia is always secondary to bronchitis.

In the so-called primary cases of bronchopneumonia the infectious agent, which is commonly the pneumococcus, starts the process by producing a sudden and violent bronchitis of the smaller bronchi, instead of producing a bronchitis of the larger tubes, which more or less gradually extends downward until the finer bronchi are involved. It is a notable fact that of all the microorganisms which produce bronchopneumonia, pneumococcus is practically the only one that also produces a lobar pneumonia, and it is also worthy of note that the younger the child the greater is the probability that a pneumococcic infection will result in bronchopneumonia rather than in lobar pneumonia.

While the inflammatory process in bronchopneumonia begins in the fine bronchi, it does not necessarily extend by continuity of surface to the associated alveoli. On the other hand, the inflammatory swelling of the mucous membrane commonly occludes the lumen of the small bronchial tubes to such an extent that we have atelectasis, or collapse of the alveoli. In the meantime, the microorganisms, exciting the inflammation, have penetrated through the small bronchi and caused inflammation of the peribronchial tissues and adjacent alveoli, while edematous tissue surrounds the collapsed alveoli above referred to. The affected lung, therefore, presents small patches of atelectasis, emphysema and consolidation. These are usually widely disseminated throughout both lungs and are surrounded by apparently normal lung tissue. As the bronchopneumonic inflammation progresses the nodules increase in number and in size, and if they happen to be in close juxtaposition they may become confluent, producing large areas of consolidation, which are indistinguishable by physical signs alone from true lobar pneumonia.

The nodules which hold in their embrace the affected bronchioles, peribronchial tissue and alveoli contain the offending microorganisms, degenerated epithelial cells, small round cells, leukocytes, and a mucoid and cellular exudate containing little fibrin. The abundant fibrinous exudate which occurs in lobar pneumonia is, as a rule, an important differentiating characteristic of the inflammatory processes in the two forms of pneumonia; yet it must be admitted that bronchopneumonia may rarely produce such an extensive and circumscribed consolidation of the lung that it is difficult even for the pathologist to differentiate the two conditions. This confusion may arise, in some instances, from the presence at the same time of both forms of pneumonia in the same or in different lungs.

**Symptomatology.**—General Symptoms.—The onset is, as a rule, gradual; there is generally a preliminary bronchitis, which grows worse, until a capillary bronchitis and a bronchopneumonia are produced. The transition, however, from ordinary bronchitis to bronchopneumonia may be more sudden; in fact, may occur over night; yet, on the whole, except in

the so-called primary form of this disease, which will be considered later, a gradual onset preceded by a preliminary bronchitis markedly distinguishes this disease from lobar pneumonia. Another distinguishing characteristic of the onset of bronchopneumonia is that the symptoms are decidedly pulmonary, pointing to a serious disease of the lungs, so that the physician is never misled, as in lobar pneumonia, into suspecting that the primary affection may be in the brain or in the abdomen.

The disease begins with a rise in *temperature,* a pronounced cough and more or less marked dyspnea. While vomiting is rare as an initial symptom, later it is quite common. With the elevation of temperature the child may complain of feeling chilly, or, if too young to describe its symptoms, the chill may be inferred by the cold extremities and the pinched expression about the face. The elevation of temperature, which marks the onset of the disease, may reach 102° or 103°F., and this is followed by remissions, the temperature continuing, as a rule, to be very irregular throughout the course of the disease, being thus in marked contrast with the sustained temperature of lobar pneumonia. The cough, which was a symptom of the preceding bronchitis, becomes more irritable and harassing, and calls unmistakable attention to the lungs as the site of the disease. If the chest of the child be now uncovered, inspection will reveal the most characteristic signs of bronchopneumonia. The *dyspnea,* which is such a marked and characteristic symptom, shows itself not only by the dilatation of the wings of the nose, but more especially by the retraction of the lower portion of the chest, where the diaphragm is attached to the chest wall. This sinking in with each inspiration of the diaphragmatic or peripneumonic groove is usually very noticeable, even in the very beginning of the disease, and, as the disease progresses, this inspiratory retraction of the chest wall becomes a very pronounced and very significant symptom, and with it there is a sinking in of the tissues in the suprasternal region. These signs, which are produced by the labor of the inspiratory muscles in their efforts to force air into the lungs, indicate an air hunger, which is caused by the closing of great numbers of small bronchi and the consequent cutting off of the alveoli from their air supply. The younger the infant the more soft and flexible are the chest walls, and therefore the more marked are these physical signs of dyspnea, which are most characteristic and significant, both from the standpoint of diagnosis and prognosis. As the disease progresses, the infant becomes more and more prostrated, the distress caused by the difficulty of breathing becomes more and more manifest, expiration is accompanied by an audible grunt, the child's expression becomes more anxious, its features drawn, and altogether it presents the appearance of being critically ill.

The *pulse rate* increases, varying from 150 to 200 per minute; in very severe cases it may be uncountable. The rapidity of the pulse, however, is not of so much importance as its character; if it be full and strong, its rapidity causes little alarm; but if it be weak, thready, intermittent, and compressible, it is an alarming symptom. From the beginning the respira-

tions are labored and increased in frequency; they may vary from 40 to 100 per minute, and with increasing dyspnea the infant may be unable to take nourishment. The prostration in these cases proceeds apace with the progress of other severe symptoms. Cyanosis is a much more marked and prominent symptom in bronchopneumonia than it is in lobar pneumonia. It not infrequently occurs early in the disease. A progressive cyanosis with coldness and blueness of the extremities is a very unfavorable sign; it indicates a weak circulation and insufficient oxygenation of the blood. If the disease gets progressively worse, all of the above symptoms become aggravated, the pulmonary distress is increased, inspiration becomes more labored, and cyanosis more marked, until the whole body has a slightly purplish appearance. The infant is no longer able to take food; it is dull, listless, and lapses into unconsciousness. The cough, which has been such a troublesome symptom, gradually grows less, and finally disappears, allowing the mucus to accumulate in the large bronchial tubes. Large râles appear in the trachea and upper bronchial tubes, the pulse becomes more feeble and flickering, the skin, especially of the extremities, grows cold, and death ensues from respiratory failure, sometimes preceded by mild convulsive movements.

If the disease in bad cases terminates in recovery, the first favorable indication noted is that the symptom group does not grow worse. If the dyspnea remains at a standstill for twenty-four or thirty-six hours, a gradual improvement may be expected thereafter. The character of the breathing from day to day becomes slightly less labored, and the cyanosis disappears. These two indications are of the very greatest importance in marking the favorable turning point in bronchopneumonia. With this improvement the temperature curve is lower, and the general condition of the patient slowly improves. The child takes food better, and is again interested in its surroundings; the harassing cough becomes more productive, as it more satisfactorily clears the bronchial tubes. This disease runs its course in from three to six weeks, and the temperature curve with its many irregularities gradually becomes normal. Bronchopneumonia, unlike lobar pneumonia, rarely terminates by crisis.

In the above outline of the onset and general clinical history of bronchopneumonia no mention is made of the physical signs elicited by percussion and auscultation. This is not because of their lack of importance from the standpoint of diagnosis, but rather because they could be better discussed as individual symptoms. It may, however, here be noted that these signs are of much less value in the diagnosis of bronchopneumonia than in lobar pneumonia. The coarse râles of a more or less general bronchitis, with perhaps small scattered areas of fine crepitation, may be heard in nearly every case of bronchopneumonia, but, after all, the diagnosis does not depend upon these findings so much as it does upon the general clinical picture above given.

Individual Symptoms.—The *fever* of ordinary bronchopneumonia is, as the accompanying charts show, very irregular. It is characterized by

marked remissions and sometimes intermissions, even when the temperature is running as high as 104° or 105°F. The remissions or intermissions usually occur in the morning, and the sharp exacerbations in the afternoon. At times the temperature may remain near the normal line for a number of days in succession, and then rise and again proceed on its irregular course.

On the whole, the temperature of bronchopneumonia is of little value from the standpoint of prognosis. A low or even a normal temperature may be present in fatal cases; this is especially true in young, malnourished infants. The temperature curve, therefore, of bronchopneumonia must be studied in connection with other symptoms. A fall of temperature,

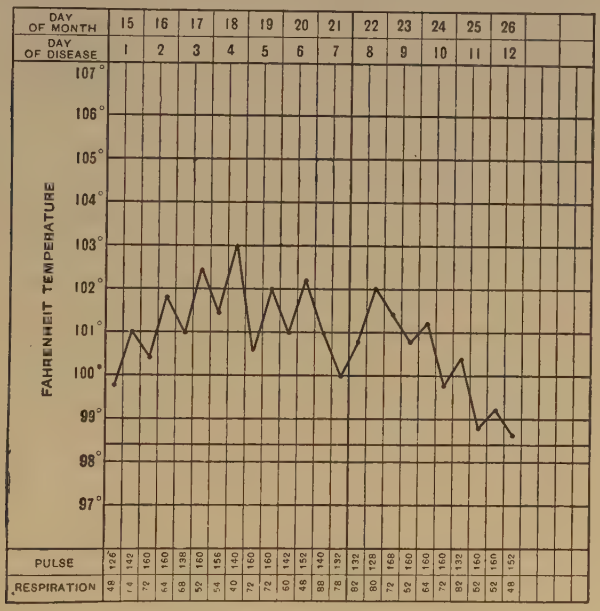



Fig. 73.—Mild Bronchopneumonia.

when coincident with an improvement in other symptoms, is a good indication, but a fall or a slow decline, when associated with no improvement, or an increase in the dyspnea and other severe symptoms, is a bad indication. A prolonged and decidedly intermittent temperature, lasting for some time, may suggest some complication, such as tuberculosis, empyema, or septic infection of the ear. In primary bronchopneumonia the temperature curve is similar to that of lobar pneumonia; it rises suddenly, remains high for from five to seven days, and then drops to normal, usually terminating by crisis.

*The Urine.*—The urine, in a large percentage of the severe cases, contains a small amount of albumin with perhaps a few hyalin and an occasional granular cast. This condition of so-called acute degeneration of

the kidney, which may occur in all febrile and toxic conditions, is of comparatively little importance. The urine clears up when the bronchopneumonia disappears, and very rarely, indeed, does acute Bright's disease develop.

*Sputum.*—The sputum, from the standpoint of diagnosis, is of comparatively little importance, because it is so difficult to obtain. In infants it is rarely, if ever, justifiable to attempt to obtain the sputum by inserting a gauze-wrapped finger into the pharynx. In older children, in whom tuberculous bronchopneumonia is suspected, this process may be successfully

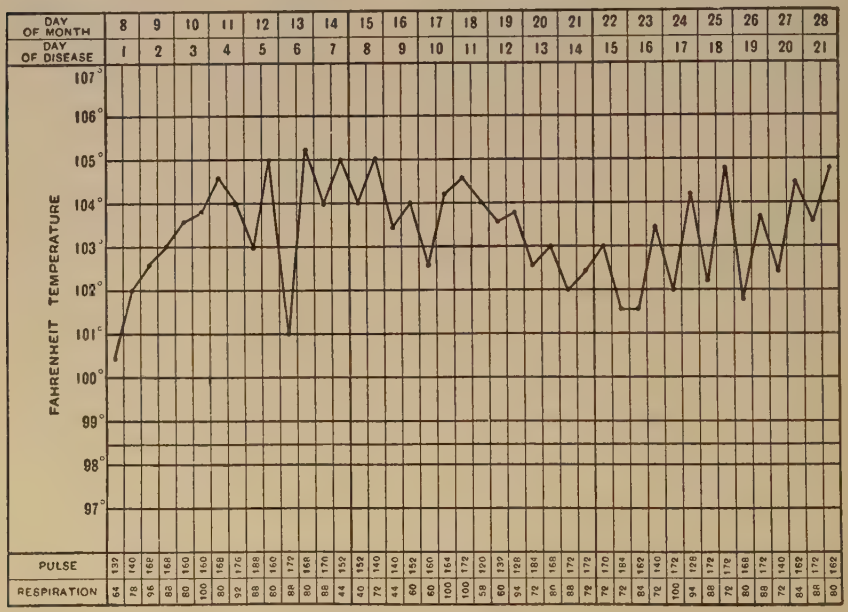



Fig. 74.—Severe Bronchopneumonia.

used, and the finding of tubercle bacilli in the sputum changes both the diagnosis and prognosis.

*Physical Signs.*—In the beginning, when small areas of consolidation are scattered widely throughout the lungs, percussion is of little value, as the note thus elicited is either normal or slightly emphysematous. Later in the disease, when these small islands of consolidation have coalesced, dullness may be elicited on light percussion; this is the more readily found posteriorly in the lower portion of the lung. At times large areas of consolidation may occur, giving rise to physical signs very similar to those of lobar pneumonia. Auscultation is of great value in confirming the diagnosis of bronchopneumonia, since in well-developed cases one can usually discover fine moist râles over small areas widely distributed, especially in the region of the spine and base of the lungs, and they may also be found anteriorly and laterally. Coarser moist râles may also be heard rather

widely distributed. The fine crepitant and subcrepitant râles found in bronchopneumonia differ from those of lobar pneumonia, in that they are more widely distributed, occurring in small areas over both lungs; they are also more evanescent, disappearing at one point and reappearing at another. As larger portions of the lung become consolidated, they may be found over larger areas and may be associated with bronchophony and bronchial breathing. The crying and coughing of the child often develop or make more pronounced the physical signs. The pulmonary inflammation is more marked in the lower portions of the lungs, and comes to the surface more readily posteriorly than it does anteriorly, because of the dorsal position of the child during its illness; as it lies so continuously upon its back the lower and posterior portions of the lungs are not properly drained.

**Types of Bronchopneumonia.**—Many variations from the ordinary type are seen. The most important of these is primary or pneumococcic bronchopneumonia. The onset and temperature curve of this condition is very similar to that of lobar pneumonia; it runs a definite course, terminates by crisis in from five to nine days, and is followed by a rapid convalescence. The general clinical picture of this condition has already been described in the chapter on Lobar Pneumonia.

An abortive type of pneumonia is recognized by all clinicians. Cases of bronchopneumonia as well as of lobar pneumonia are seen, which begin with the typical onset and show the characteristic symptoms and physical signs of these diseases, in which suddenly, on the third or fourth day, the temperature falls to normal, the general symptoms as well as the physical signs disappear, and a rapid convalescence follows. These cases are described by some authors under the term "Acute Pulmonary Congestion," but there is ample clinical and pathological proof that many of these cases are pneumonia.

Prolonged bronchopneumonia with migrating areas of consolidation is commonly produced by the influenza bacillus.

Bronchopneumonia of the New-born.—During the first weeks of life bronchopneumonia is a very insidious disease, and usually occurs as the sequel of epidemic grippe or influenza. The earliest symptoms are cold in the head, nasal catarrh, slight cough, and pharyngeal irritation, followed perhaps by laryngitis, bronchitis, and finally bronchopneumonia. The progress of the disease, however, in the infant is so masked that it is likely to be overlooked. There is but slight elevation of temperature associated with an increasing cough and slight dyspnea, and as the infant of this age has so little resistance, a well-marked and incurable bronchopneumonia not uncommonly develops before serious disease of the lung is suspected. At this age the disease is nearly always associated with gastrointestinal disturbances, which hasten its unfavorable termination. Although this condition is comparatively rare and is most commonly seen in weaklings, yet the fact that it may develop so insidiously should make the physician most careful to give prompt attention to all catarrhal diseases

of the upper air passages in young infants. Little can be done to save them after the bronchopneumonia has developed, but much can be done to safeguard them against this disease if the early catarrhal conditions are properly looked after.

Deglutition Pneumonia.—Following intubation, and paralysis of the soft palate, food particles infected in the diphtheritic throat may be aspirated into the lung and cause pneumonia. It is not the foreign body but the infection it carries with it that causes the localized inflammation of the lung. In whooping-cough the deep inspiration which follows the paroxysm of cough may suck particles of food far into the branches of the bronchial tree. If these foreign particles are infected and become lodged, they may excite a localized bronchopneumonia, terminating in abscess. Noninfected food particles or other clean foreign bodies may cause bronchial irritation, but not bronchopneumonia.

Bronchopneumonia Following Gastroenteritis.—One of the dangers of chronic and subacute enteritis is a complicating bronchopneumonia. In many of these cases it is a terminal infection, occurring but a short time before the fatal issue. Terminal bronchopneumonia may also occur in other wasting diseases.

Tuberculous Bronchopneumonia.—This is one of the common forms. In the beginning it presents the picture of an ordinary bronchopneumonia, but fails to clear up in three or four weeks, although the temperature and acute symptoms may have somewhat subsided. It occurs most frequently in infancy, but may occur in older children. The cough is tenacious, the dyspnea is marked, although not excessive, the fever is remittent, sometimes intermittent, falling in the morning and rising to 104° or 105°F. in the afternoon. The sharp exacerbation in temperature which occurs at least once in twenty-four hours does not always come at the same time of the day. There is progressive emaciation and loss of strength, and the disease may continue for five or six weeks. As it progresses, large areas of consolidation appear; the whole of both lobes posteriorly may be consolidated, or the consolidation may be in other parts of the lung. Over these consolidated areas there is marked dullness, tubular breathing and bronchophony, and large and fine râles may be heard. The physical signs are, in fact, almost identical with those of lobar pneumonia. The diagnosis may sometimes be made by examining the sputum. As a rule, there is a family history of tuberculosis, with perhaps a previous personal history of glandular tuberculosis. The areas of consolidation are persistent and do not shift, as in other forms of prolonged bronchopneumonia.

**Complications.**—Pleurisy is as important, although not so common, a complication of bronchopneumonia as it is of lobar pneumonia. When it does supervene, however, it is usually purulent in character. An empyema complicating a bronchopneumonia may prolong the fever and cause the temperature curve to assume a septic type, falling to normal or below normal in the morning and rising rapidly to 104° or 105°F. in the afternoon; it does not always reach its highest or its lowest point at the same

hours every day, and there may be more than one such exacerbation during the twenty-four hours. This irregular, sharply remittent or intermittent fever, following a bronchopneumonia, usually indicates either empyema, otitis media, or a complicating tuberculosis. The differential diagnosis between these conditions must be made by the associated symptoms. Otitis media is a frequent complication. It may produce the septic temperature curve above described, and under these conditions, if empyema and tuberculosis can be excluded, the existence of an otitis media becomes more probable. Moreover, it is usually associated with pain, so that the increased restlessness, sleeplessness and irritability of the child may suggest to the physician the possibility of earache, and an examination of the ear drum may confirm the diagnosis. Not infrequently, however, this complication is overlooked until the mother or nurse announces that the patient has a purulent discharge from the ear. Pericarditis is a rare complication. When it does occur, however, it is nearly always purulent and leads to a fatal issue. A weak and dilated heart muscle may be an unfavorable complication in cases of bronchopneumonia, associated with severe attacks of whooping-cough. Meningitis, arthritis and osteomyelitis are possible complications.

**Diagnosis.**—In lobar pneumonia, as previously noted, it is often a question of the organ involved, as the symptom group in this disease may be so misleading that the physician's attention is not called to the lung as the site of the disease. This very rarely occurs in bronchopneumonia. In this disease the pulmonary symptoms are so prominent that attention is at once directed to the lungs. The only question, therefore, which is likely to arise is as to the character of the pneumonia. Is it a lobar, or a bronchopneumonia? If the latter, what is the character of the bronchopneumonia? As the differential diagnosis of the different forms of bronchopneumonia have already been considered, it only remains here to note the points of difference between lobar and bronchopneumonia. When the characteristic physical signs of these diseases are well defined there is little difficulty. In lobar pneumonia we may have large areas of consolidation confined to one lobe, with marked dullness, bronchial breathing, and subcrepitant râles scattered over this consolidated area, the other portions of the lungs being comparatively free from physical signs. In bronchopneumonia the dullness may be absent, but fine crepitant and subcrepitant râles may be found from time to time in small areas scattered rather widely over both lungs, and with this we have coarser râles in the larger bronchi. Unfortunately, however, these clearly defined physical signs are not always present, or they may be so commingled in an individual case that the physician is left in doubt as to the character of the pneumonia. For these reasons the clinical history of the two pneumonias is of quite as much importance as the physical signs in making a diagnosis. Bronchopneumonia differs from croupous pneumonia in that it is usually a secondary disease, having a more gradual onset. Its temperature curve is not at all characteristic, being irregular in character and running no definite course, and,

most important of all, dyspnea and possibly cyanosis may be marked and early symptoms. The early and more or less characteristic dyspnea, which has above been carefully dwelt upon, is of great value in differential diagnosis.

**Prognosis.**—This is one of the most serious and dangerous diseases of early life. Even under the most favorable conditions the death rate is 25 per cent. In hospitals and other institutions, where children are crowded together, the mortality may reach from 40 to 60 per cent. Age is an important factor in determining the death rate. In the new-born the disease is almost always fatal; during the first year of life it reaches 30 to 40 per cent.; during the second year of life it falls below 25 per cent., and thereafter continues to diminish, until between the third and fifth year of life the mortality, under favorable conditions, generally does not exceed 10 per cent. The physical condition of the child greatly influences the death rate. The mortality is very high in syphilitic, rachitic, tuberculous, and other malnourished infants, and when associated with enteritis is almost always fatal. The mortality is much greater in bottle-fed than in breast-fed infants. The death rate is higher in the middle and northern States during the cold and changeable winter months, because this climate and this season are not so favorable to the fresh-air treatment of this disease. The mortality is also influenced by the character of the infection which produces the bronchopneumonia. In tuberculous cases it is bad. In pneumococcic or primary bronchopneumonia the prognosis is good; nearly all of these cases get well, except those which occur in weak and delicate infants during the first year of life. The mortality is higher in the cases following diphtheria, scarlet fever and whooping-cough than it is in those produced by measles, influenza or simple bronchitis.

**Prophylaxis.**—Fresh air and good hygiene are the keynotes in the prophylactic treatment of this disease. Bronchopneumonia, in the vast majority of cases, is secondary to bronchitis, and there is little doubt that if children suffering from the various acute infectious diseases and from ordinary simple bronchitis could be isolated from other children, and could have fresh, pure air, uncontaminated by bacteria, bronchopneumonia would be a comparatively rare disease. Many of the cases of bronchitis occurring in institutions, and in tenement houses, are forced to breathe impure air, which carries secondary infections of various kinds to their bronchial mucous membranes, and bronchopneumonia results. The necessity, therefore, of looking upon every case of bronchitis in infancy with reference to the possible development of bronchopneumonia is of the greatest importance. Every case should be isolated, protected from all possible contagion, and, above all, should have plenty of fresh air. The younger the child the more important are these precautions. In infants, during the first few weeks of life, every simple coryza and pharyngeal irritation should receive prompt and careful attention, since at this age the prophylactic treatment of bronchopneumonia is the only treatment that is of any avail.

**Treatment.**—The first and most important thing in the treatment of

bronchopneumonia is to place the patient under proper hygienic surroundings. A large, well-ventilated and isolated room is to be selected, in which the patient is to remain under the care of a competent nurse. All unnecessary callers are to be excluded, in order that quiet may reign, and that the air may be as little contaminated as possible.

Hygienic Treatment.—Fresh air is the most important curative agent in bronchopneumonia, even more important here than in lobar pneumonia. At all seasons of the year the windows of the room must be open, to give an abundant inflow of fresh, cool air. The importance of the fresh-air treatment of this disease cannot be exaggerated, and the laity must be given to understand that without it all other methods of treatment are comparatively useless. For many years Northrup has taught and has insisted that the fresh-air treatment would greatly reduce the mortality of this disease, and he has done much, as have thousands of other physicians, to bring the laity to understand that there is little or no danger of "catching cold" by the fresh-air or outdoor treatment of bronchopneumonia, provided it is properly carried out. What the laity calls "catching cold" results from exposing these patients indoors to an air that is contaminated with pathogenic microorganisms. Fresh, pure, cold air must, therefore, be had wherever the patient is located. The outdoor air of any locality is infinitely better than the indoor air; so that if these cases must be treated in the downtown, smoky atmosphere of our closely crowded tenement houses, the windows should be opened and this air let in in abundance, to replace the contaminated air of the sick room. During winter the body of the child must be kept warm with proper clothing, and possibly artificial heat. At this season the infant should always be carefully hooded with some warm material, and the temperature of the room in the neighborhood of the child's bed should not be allowed to fall below 60° or 65°F. This can be accomplished by artificial heat, even though the end and side windows of the room be open. The bed of the infant is not to be placed between the windows.

Dietetic Treatment.—From what has been said of the seriousness of gastrointestinal complications, one realizes the importance of the dietetic treatment. If the child be a nursing infant, breast feeding must be persevered in; and as the child becomes too ill to take the breast, as it almost always does within a few days, every effort must be made with breast pumps and other devices to keep the mother's milk from drying up, and to secure in this artificial way a certain amount of breast milk, which can be fed to the infant with a spoon. In artificially fed babies the food selected must depend upon the age and digestive capacity of the individual infant, the physician remembering that whatever may have been the digestive capacity of the baby during health, it is perhaps diminished one-half by an attack of bronchopneumonia. In young infants, therefore, the milk formula given in health should be reduced not only in strength, but in quantity. Predigested foods, skimmed milk, meat juice, and albumin water may be necessary in the feeding of young and delicate infants. The

importance of this subject is so great that the clinician should understand that, especially in early infancy, it is just as important not to overfeed as it is to give foods which are within the digestive capacity of the individual infant.

Whiskey or brandy should be given in all cases of bronchopneumonia. Of the two, good old rye whiskey is perhaps the better. It may be given well diluted with water, and sweetened if necessary. Most children can be induced to take it in this form without resistance. In a long-continued disease, such as bronchopneumonia, where the child is necessarily underfed, whiskey serves as a food; it keeps up the strength and prevents excessive waste of tissues. An infant one year of age may take 20 or 30 drops every four hours; a child two or three years of age, a teaspoonful. Larger or stimulating doses of whiskey or brandy may be given in bad cases, especially when the amount of food taken is small. I have for many years used whiskey as a routine measure in all severe cases of bronchopneumonia, and have found that it rarely, if ever, produces gastrointestinal disturbance.

Medical Treatment.—In beginning the treatment the bowels should be opened with castor oil, and this should be repeated every third or fourth day throughout the disease. This serves the purpose of carrying off the mucus which the child has coughed up and swallowed, and perhaps thereby prevents intestinal infection.

Tincture of strophanthus or tincture of digitalis (the former is preferable) should be given every four hours throughout the course of the disease. One drop of tincture of strophanthus is a suitable dose for a child one year of age, and two drops for a child three years of age. Caffein-sodium-benzoate, or salicylate, in one-grain doses by the mouth, or one-half-grain doses hypodermically, is a valuable circulatory stimulant, which may be given if the pulse becomes feeble and intermittent.

Strychnin is another drug very widely recommended, and is of value both as a general tonic and as a respiratory stimulant. It may be given combined with whiskey, 1/300 of a grain every three or four hours, to an infant one year of age, and 1/200 of a grain to an infant two years of age. The chief objection to the use of strychnin is that its bitterness makes the whiskey unpalatable, and for this reason it often becomes necessary to force the infant to take this combination. It is a wise policy, and one that should be followed within limits, to cater to the tastes of infants both in food and medicines when they are seriously ill with any disease. It may, therefore, be unwise to attempt to mix with the whiskey unpalatable medicines which will cause the infant to struggle against their administration. Forcing either foods or medicines into the stomach of a child very ill with bronchopneumonia not only exhausts the strength, but often causes vomiting. The use of strychnin, therefore, is perhaps better restricted to the later stages of the disease, when a respiratory stimulant is urgently needed and should then be given hypodermically. Oxygen is a good respiratory stimulant, and before the days of the fresh-air treatment was one of the most valuable remedies. It still, however, has a place in the

treatment of this disease, even when the child is getting all the fresh air it can possibly have. It is especially indicated in the later stages when the inspiratory dyspnea is very marked and cyanosis is present. It is easily administered by inhalation without disturbing the child; the funnel which is connected with a tank of oxygen by rubber tubing is suspended just above its mouth and nose.

Sedatives must be used with great care in bronchopneumonia, and they are of much less value here than in lobar pneumonia. The temptation is very great to try to influence with sedatives the irritable and harassing cough of bronchopneumonia. But it should be remembered that the younger the child the more dangerous is all sedative medication. Opiates are rarely indicated under two years of age. Now and then, perhaps, a sturdy infant with a severe and harassing cough may be slightly benefited by a few drops of paregoric or a small dose of codein. But within the last ten years I have not in a single case thought it advisable to give opium in any form to a patient under two years of age. Opium produces constipation, destroys the appetite, disturbs the digestion and does much more harm than good. I believe that the injudicious use of opium as a cough sedative has in the past been responsible for no small percentage of the deaths produced by bronchopneumonia. Opium, therefore, should, with the expectorants and medical antipyretics, be classed among the dangerous rather than the beneficent remedies in bronchopneumonia. Bromid of potash in four- to five-grain doses, with tincture of belladonna in one-minim doses, may, when combined in some palatable vehicle, serve a useful purpose as a cough sedative in certain cases, but even these drugs find their greatest indication in children over two years of age. Under this age they should be used only when absolutely necessary and should be discontinued if they produce the slightest gastric or intestinal disturbance.

Expectorants should have no place in the treatment of bronchopneumonia in children under two years of age. Tartar emetic, syrup of ipecac, syrup of squill, carbonate and muriate of ammonia, in my opinion, do more harm than good. The widespread use of these drugs is, I believe, responsible for no small part of the mortality of this disease in early infancy. They may dislodge a certain amount of mucus in the throat and upper air passages, but the temporary improvement in the breathing, produced in this way, is more than counterbalanced by the harm they do in destroying the appetite, disturbing the digestion, and cutting off the nutrition of the child. In a long and prostrating disease, such as bronchopneumonia, any medicine that interferes with nutrition or disturbs the appetite or digestion will do more harm than good. In older children the careful use of these expectorants may perhaps be of value.

Antipyretics are of much less value and are much less frequently called for in bronchopneumonia than in lobar pneumonia. The fever of bronchopneumonia is remittent or intermittent in type, and little or nothing is to be gained by drugs or other agents used for lowering the temperature. Phenacetin, antipyrin, and other drugs of this class, which may occasionally

be used to advantage in older children suffering from lobar pneumonia, do much more harm than good in bronchopneumonia. Cold packs, which are of value in many cases of lobar pneumonia, are not so generally used in bronchopneumonia, and they are, in my opinion, decidedly contraindicated in infants suffering from marked inspiratory dyspnea or cyanosis. A warm bath twice a day, or a tepid sponge bath three or four times in twenty-four hours, is of value. These baths act as a sedative to the nervous system, promote elimination through the skin, and serve as a general tonic to nutritional processes. They are not given with the idea of lowering the temperature. Priessnitz applications are of value in both forms of pneumonia; a light, sleeveless flannel jacket made to fit the child is dipped in and wrung out of water at a temperature of 70°F.; this is snugly applied to the entire chest and covered with a similar dry flannel jacket. The wet jacket may be removed and reapplied at intervals of from ½ hour to 3 hours, as the exigencies of the individual case demand.

Counter-irritants and Poultices.—Nearly all writers, at the present time, recommend counter-irritants and condemn poultices. Notwithstanding this almost universally expressed opinion, I believe that the present-day teachings are too liberal in their recommendation of counter-irritants, and too sweeping in their condemnation of poultices. Counter-irritation is of much less value in bronchopneumonia than it is in lobar pneumonia. If a counter-irritant is applied to the chest of an infant suffering from bronchopneumonia, it must, to do any good, cover the skin of the entire chest, or be applied with special severity to the skin covering its posterior surface. Counter-irritations with mustard plasters and mustard baths frequently do more harm than good; to be of any value they must redden the skin and produce more or less discomfort and irritation, and this increases the child's restlessness, nervousness and sleeplessness, without perhaps producing any favorable influence on the general and widespread inflammation of the lungs, which is seated some distance beneath the skin. If counter-irritation of any kind is used, I much prefer warm camphorated oil of double strength. When this is rubbed into the chest of the child it produces a mild counter-irritation, and the camphor, some of which is perhaps absorbed, acts as a general stimulant. On the other hand, poultices and the oil-silk jacket so universally condemned are of great value in the early stages of bronchopneumonia when the disease is spreading, or when it is passing over from a general bronchitis into a capillary bronchitis, or bronchopneumonia. I believe that a thin, light, warm flaxseed poultice spread over the back and chest of the child and covered with oil-silk, is a remedy of the very greatest value in preventing the extension of this disease. Poultices, when properly used in connection with the open-air treatment, do not make the child uncomfortable or increase its temperature; on the other hand, they are sedative, rather than irritating, but they can be successfully managed only when the patient is under the care of nurses who understand how to make and how to apply them without unnecessarily exposing the infant to draughts of cold air. It has been my

practice to change the poultices at intervals of two hours, and when this is done the patient is carried for a few minutes from the cold room into an adjoining room, and as soon as the poultice is adjusted he is immediately returned to his fresh-air chamber. The poultice is of especial value in the very onset of bronchopneumonia, and is to be used only during the time the disease is progressing. The oil-silk jacket lined with a thin layer of cotton-wool may be used where the poultice cannot be satisfactorily handled, and it may be substituted for the poultice after its discontinuance. After many years of experience with the oil-silk jacket and the poultice, I am to-day a firm believer in their efficacy, and think they are the most important agents we have for stopping the spread of bronchopneumonia in its early stages. I fail to see what possible harm they can do when combined with the open-air treatment of this disease. I have, in connection with the oil-silk jacket, for many years used the following prescription, which is to be applied as an inunction to the chest of the child twice in twenty-four hours:

| | |
|---|---|
| Guaiacoli | ʒ i |
| Lanolini anhydrous | q. s. ad ℥ i |

One-half level teaspoonful applied as an inunction to the chest twice a day.

This guaiacol-lanolin prescription, when well rubbed in, is readily absorbed, the guaiacol appearing in the urine within two hours after its application. The guaiacol thus administered, while it may never reach the pulmonary mucous membrane, certainly acts as a lymphatic antiseptic, and as it passes from the skin through the lymphatics it may favorably influence the lymphatic involvement which always occurs in bronchopneumonia. This drug, administered in this way, is of especial value in those cases complicated by lymphatic or pulmonary tuberculosis.

Treatment During Convalescence.—Following the disappearance of the acute symptoms, there is usually a period of slow convalescence; the child is weak, nervous and anemic. During this time the fresh-air treatment is to be continued, and a carefully selected diet suitable to the nutritional demands of the child prescribed. One of the thick malt extracts combined with cod-liver oil or iron, or some form of arsenic, or syrup of the iodid of iron, or hydriodic acid, may be valuable tonics during this period. In children predisposed to tuberculosis creosote, or the benzoate or carbonate of guaiacol may be given with benefit.

## CHAPTER LIII

### PLEURISY

Pleurisy is an inflammation of the pleura, usually secondary to infection elsewhere, most commonly in the lungs. This inflammation may occur either without or with effusion into the pleural cavity. The former is called dry pleurisy. The latter occurs in two forms, the serofibrinous and purulent, depending upon the character of the exudate; these are frequently commingled, the case beginning as a serous and later becoming a purulent pleurisy. The dry form is not so infrequent as clinical records would indicate; it is commonly overlooked, being masked by the accompanying pneumonia, or other causative disease. The purulent form (empyema) is both relatively and actually much more common in the child than in the adult.

**Etiology.**—Infection with pathogenic microorganisms is the cause of this disease. In infancy and childhood the pneumococcus is the common cause; this is especially true of the purulent form (empyema). Koplik found this organism in 75 per cent. of his cases; Netter, Beck, and other investigators report similar findings. The percentage of cases due to the pneumococcus is greatest in infancy, and slowly diminishes with advancing childhood. Infantile empyema is nearly always due to this organism, while in the adult only about 25 per cent. of the cases are due to this cause. Streptococci and staphylococci are the next most common organisms associated with pleurisy; these cases usually occur as septic complications of the acute infectious diseases; they are not as common in the infant and young child as in the older child and adult. Of special interest is the fact that tubercle bacilli are much less frequently a cause of pleurisy in the infant and young child than in the adult. Tuberculous pleurisy is a comparatively common disease in adult life, while in childhood only about 4 or 5 per cent. of the cases are due to this cause. In a fair percentage of tuberculous pleurisies neither the tubercle bacillus nor other microorganisms can be demonstrated in the fluid. Negative findings in the purulent exudate are suggestive of tuberculosis, but even when these negative cases are included the percentage of tuberculous pleurisies remains as low as above stated. Other microorganisms, such as the typhoid, colon, and influenza bacilli, may produce pleurisy, but these cases are relatively rare.

Pleurisy may be a primary disease, but in the vast majority of the cases it is secondary. The primary cases are, usually, the first manifestations of a rheumatic or pneumococcic infection. The secondary cases, for the most part, are associated with or occur as complications of lobar pneumonia, bronchopneumonia, or acute bronchitis. The various acute infectious diseases, especially rheumatism, influenza, scarlet fever, diphtheria, follicular tonsillitis, measles, whooping-cough, typhoid fever, tuberculosis,

chronic gastroenteritis and septicopyemia, may produce pleurisy. In the new-born sepsis is the most important cause.

Pleurisy is most commonly seen between the sixth month and the sixth year, and occurs with diminishing frequency before and after this period. The serofibrinous type is occasionally observed in the infant, becomes more common after the third year, and occurs with increasing frequency from this time on, so that in later childhood and adult life it is much more common than purulent pleurisy.

Pleurisy occurs more frequently in boys than in girls, and is much more prevalent during the cold, damp months of winter and spring than it is in the warmer and dryer months. Exposure to damp, cold weather or "catching cold" is perhaps an important exciting cause, which can act, however, only by producing a more favorable soil for the microorganisms which cause this disease.

**Pathology.**—On post-mortem examination old pleural adhesions, with more or less marked thickening of the pleura, may be found in children dying of other diseases; such unsuspected lesions commonly result from the dry or fibrinous form of pleurisy. In this form, during the acute stage, there is found on the congested, inflamed, and thickened pleura a fibrinoplastic exudate, with perhaps a slight amount of yellow serum in the pleural cavity. The rubbing together of these roughened surfaces produces the characteristic friction rub. The effusion and exudate in these cases are absorbed, leaving the pleural surfaces adherent or bound together by fibrinous bands at certain points. These adhesions, however, produce little or no damage, since they do not to any extent interfere with lung expansion.

Pleurisy with effusion presents a very different pathological picture. Whatever may be the character of the effusion, the lung is pressed upward until there is very little expansion on the affected side. The pleural cavity in these cases is filled with serous, seropurulent or purulent fluid, which, in a small percentage of cases, is tinged with blood. Not infrequently this exudate is encapsulated and thereby separated from the rest of the pleural cavity, or in rare instances more than one encapsulated sac of fluid may be held between the pleural surfaces, so that the tapping of one of these cavities does not reach the other. In properly treated cases, especially those in which the fluid is serous, complete disappearance of the effusion may be obtained with comparatively little damage to the pleural cavity, and with little or no diminution of the respiratory capacity on the affected side. The lung in these cases refills the chest cavity, and the resulting pleural adhesions have little or no influence in impeding respiratory movements. In other cases, more especially in empyema, there is great danger that extensive pleural adhesions will not only obliterate the pleural cavity, but that the lung on the affected side may remain in a state of partial shrinkage due to the inflammatory adhesions. In such instances the respiratory capacity of the lung may be greatly diminished and the resultant deformity of the chest and spine be very great. Un-

treated cases of empyema may ulcerate either through the parietal or visceral pleura, on the one hand producing a subcutaneous abscess, or on the other discharging the pus into the bronchial tubes. The quantity of fluid may reach from 1,000 to 4,000 c. c.

**Symptomatology.**—General Symptoms.—Pleurisy is commonly manifested by fever, cough, pain in the chest, disturbances of respiration and rapid pulse. Any or all of these symptoms may be absent in individual cases, but on the whole, in well-marked pleurisy, especially where there is an effusion in the pleural cavity, this symptom group in whole or part is present, and with it headache, vomiting and constipation are not infrequently associated.

In the most common group of cases, those following lobar or bronchopneumonia, the pneumonia usually runs its course with its characteristic symptoms, but following the fall in temperature between the seventh and the tenth day the patient fails to convalesce, there are a secondary rise of temperature, an aggravation of the cough, sharp pain in the chest, increasing dyspnea, rapid pulse, and a careful physical examination reveals a beginning pleurisy. In other instances the pleurisy supervenes during the pneumonia, and may become at once the dominant symptom group; in these cases the prolongation of the fever with the cough, dyspnea, pain in the chest and rapid pulse are important symptoms indicating this complication. The fever may reach 104° or 105°F., and the pulse may run above 160. In another group of cases, much less common, the pleurisy appears as a primary disease; in this variety the patient is taken suddenly ill with headache, vomiting, and chilliness, and the fever rises to 102° or 103°F., and the hacking cough, pain in the chest and shallow, rapid breathing quickly develop. In still another group of cases the disease is not announced by acute symptoms; the child for a week or ten days is languid, has perhaps a slight cough, little or no appetite, gradually loses strength, becomes more or less anemic, has a slight intermittent fever, with more or less marked night sweats, and during this time, while it is clearly evident that the child is ill, there may be little to call attention to the fact that he has a well-marked pleurisy with an effusion in the pleural cavity. The character and even the location of the disease in such cases are finally revealed by physical examination of the chest.

Individual Symptoms.—The *fever* in the serofibrinous form, while it may be high in the beginning, quickly subsides and runs an irregular course, reaching 101° or 102° F. Under proper treatment it usually becomes normal within a week or ten days. In empyema the fever is irregularly intermittent or remittent, rising to 104° or 105°F., and frequently falls to normal or below normal; when due to pneumococcic infection the variations in temperature are not so marked, but when due to sepsis following the acute exanthemata it is characterized by rapid rises and falls, and is commonly associated with sweating. Following drainage of the pleural cavity, it should run a mildly intermittent course until convalescence is established.

*Pain* in the side of the chest aggravated by coughing and by deep inspirations, is one of the most significant and valuable symptoms. It calls attention to the location and nature of the disease. The "stitch" in the side is an early symptom which usually disappears when the effusion has increased sufficiently to have separated the pleural surfaces. It should also be remembered that in young children the pain caused by coughing and inspiration is not infrequently referred to the abdomen.

The *cough* is irritating, dry, hacking and painful, and the patient makes an effort to suppress it. The anxious, worried, and pained expression which spreads over the face of the child when it feels it can no longer suppress the cough is very suggestive of pleurisy. With the appearance of the effusion, the cough may become less painful and less frequent.

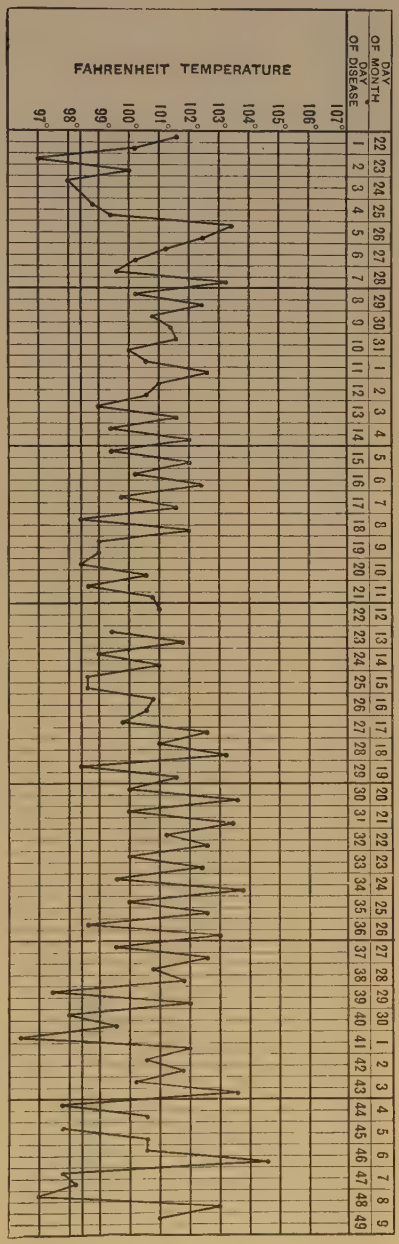



Fig. 75.—Empyema Following Pneumonia.

*Respiration* is more or less painful, and the child breathes superficially so as to limit the chest expansion. The respiratory movements are rapid and are accompanied by grunting; dyspnea is usually a gradually increasing symptom, which after a time becomes very marked. In its efforts to suppress respiratory movements on the diseased side the child lies on the affected side, and makes the unaffected lung do as much of the work as possible; this attitude is most suggestive and characteristic. As the pleural cavity fills, the respiratory movements are less painful, but the dyspnea and rapid breathing are more marked.

In left-sided pleurisy with effusion, the heart is displaced and impeded in its action by the accumulating fluid. In these cases the pulse, which is at all times rapid, may reach

150 to 180, and is commonly feeble. In severe cases cyanosis is present. With the progress of the disease there is more or less marked facial pallor,

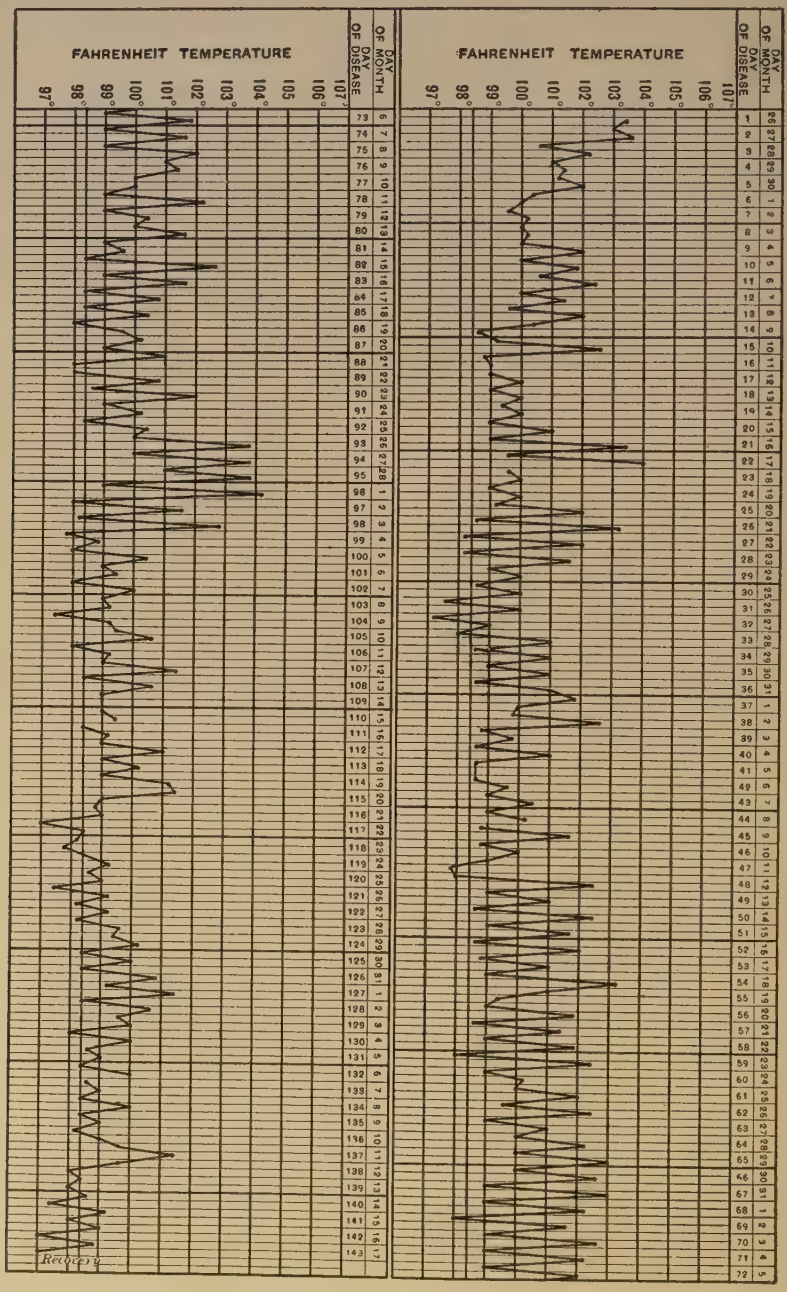



Fig. 76.—Empyema.

especially about the lips, and the child gradually grows more anemic and emaciated.

In pleurisy with effusion the following points are revealed by *physical examination*: Inspection shows diminished respiratory movements on the affected side, and this side of the chest appears larger and the intercostal spaces are less evident. Palpation may show that the vocal fremitus is diminished or lost over that portion of the chest which is filled with fluid; in extensive effusions it cannot be felt over the whole of the lower portion of the chest cavity. Palpation also reveals the diminished respiratory movement of the affected side and the increased movements on the well side. In left-sided pleurisy it reveals the displaced apex beat, and in right-sided pleurisy the lower border of the liver may be felt pushed somewhat downward into the abdominal cavity. Percussion gives the most valuable information. With the child sitting in an upright position one can, as a rule, outline the fluid by the dullness, which in most instances amounts to flatness, over that portion of the chest cavity filled with fluid. Directly above the line of dullness the resonant note of the lung, which may be almost tympanitic, is elicited. In many instances the upper line of dullness shifts with the position of the child, being affected by gravitation of the fluid. From the standpoint of diagnosis the peculiar resistance, which is felt by the percussing finger, is second only in importance to the dullness or flatness obtained by percussion. These two signs rank all others in value in the diagnosis of pleurisy, and they alone justify the introduction of an exploring needle to ascertain not only the presence, but the character, of the pleural effusion. It should be remembered that, while the flatness elicited by percussion is commonly found in the lower portion of the chest, especially posteriorly, it may also be found in other parts of the chest, being there produced by encapsulated fluid, and that the dullness elicited over these encapsulated areas may not amount to absolute flatness. In some instances, especially when the encapsulated fluid exists along the back of the lung, one may on deep percussion obtain a slightly resonant note from the lung situated beyond the fluid. Attention should also be directed to the fact that where the pleural cavity is filled with fluid, and the lung is pressed upward and forward, a very resonant tympanitic note may be obtained at the apex anteriorly, while all the remaining portion of the chest is flat on percussion and gives a peculiar board-like resistance to the percussing finger. In these cases of extensive effusion the dullness extends beyond the opposite border of the sternum. Auscultation is of much less value than percussion. Early in the disease, however, one may be able to hear the characteristic friction rub which coincides with inspiration, or expiration, or with both. The to-and-fro friction rub, when it can be heard, is a sign of great value. It disappears early with the increase of effusion and is heard again more distinctly when the fluid almost or quite disappears. Over the fluid the respiratory sounds are absent, indistinct, or distant, but on the whole the auscultatory findings in pleurisy with effusion are very unsatisfactory and at times misleading. This applies especially to infants and young children. Bronchial breathing, bronchophony, and even respiratory sounds may be heard over areas containing fluid.

*Displacement of Other Organs.*—In left-sided pleurisy with effusion the displacement of the apex beat of the heart toward the median line is one of the most valuable findings; it may be pushed over as far as the sternum. In right-sided pleurisy the displacement of the liver downward, when it can be clearly demonstrated, is a valuable sign.

Fluoroscopic examinations or X-ray pictures may locate the fluid, show displacement of the heart and other organs and thereby give valuable diagnostic information.

Fig. 77.—Pleural Effusion in Left Side of the Chest.

*Exploratory Puncture.*—The ultimate diagnosis and also the prognosis will largely depend on the results of an exploratory puncture. This should be made at a point over the area of greatest dullness and where there is an almost or complete absence of fremitus. In cases where the flatness is extensive the point chosen should be over the lower portion of this area. In the average case, where the whole lower portion of the pleural cavity is filled with fluid, the point of selection should be the posterior axillary line, at the sixth interspace on the left side and the fifth interspace on the right.

In exceptional cases encapsulated fluid will be found over other areas, and the lower portion of the pleural cavity will be free from fluid, so that the physical examination must in every case determine the point for introducing the needle. The child is firmly held in a sitting position, the skin is thoroughly cleansed, and a clean needle, attached to a syringe, is introduced from 1 to 2 cm. until it reaches the pleural cavity. The piston is then gently drawn and the fluid, if it be reached by the needle, will flow into the syringe. If fluid is not obtained it should not be sought for by moving the point of the needle in the pleural cavity, but it should be immediately withdrawn; after a second careful physical examination, to again determine the point of greatest flatness, the skin and needle should again be cleaned and the needle introduced in this place in the hope of striking fluid. In making this exploratory puncture a needle of fair size (one millimeter) should be used, as a smaller one is likely to become clogged by pus or fibrin. It should pass well under the border of the rib to avoid injuring the intercostal artery which runs along the inner and lower border of the rib. The wound left by the introduction of the needle should be immediately covered with adhesive plaster. There is almost no danger from an exploratory puncture when it is made through a clean skin with a sterile needle unless it be pushed through the pleural cavity into an infected lung, and this danger is rather remote. It is also important to avoid the cardiac region as much as possible so as not to wound the pericardium. The character of the pleurisy is determined by the fluid obtained by this exploratory puncture; it may be serous, serofibrinous, seropurulent, or purulent in character and may contain blood. A careful bacteriological examination of the fluid thus obtained may determine the character of the infection. If the bacteriological examination shows the pneumococcus to be the dominant or sole microorganism present, the prognosis is better and the disease will run a milder course than in those cases in which streptococci and staphylococci are found in abundance. Tubercle bacilli are rarely found in the fluid, not only because tuberculous pleurisy is comparatively rare in childhood, but also because even in cases due to tuberculosis the tubercle bacilli are not readily found. Inoculation experiments with guinea-pigs may be resorted to in chronic cases in which tuberculosis is suspected. If the fluid be of light yellow color and serous in character the probabilities are that the disease will remain a serous pleurisy; this inference is especially true in children over five years of age, since, at this time of life, this form of pleurisy is much more common. If, however, the serous fluid is found on microscopical examination to contain pneumococci, streptococci, or staphylococci, and if, at the same time, it contains a considerable number of lymphocytes and pus cells, the probabilities are that this serous pleurisy will in a short time be converted into an empyema; this inference is especially true in young children under five years of age, since at this period purulent pleurisy is the most common form. The effusion, both in serous and purulent pleurisy, may be tinged with blood, but this is of little diagnostic

or prognostic importance. In the adult, blood is an unfavorable sign, since it commonly means tuberculous pleurisy, but this is not true in the child. At this age tuberculous pleurisy may occur without the fluid being tinged with blood, and again the effusion may be bloody in other forms of pleurisy. The presence of blood therefore in the young child is of little importance unless it occurs in low forms of pleurisy associated with hemorrhagic diseases, such as scurvy.

**Treatment.**—The fresh-air treatment is quite as important in pleurisy as it is in pneumonia; apart from this, the disease should be treated symptomatically. In serous pleurisy, or in those cases in which the character of the fluid has not been determined, the treatment for the time being is largely expectant. The patient is put to bed, and, if he be under two years of age, his diet should be liquid in character and carefully selected to suit his diminished digestive capacity. In older children suffering from serous pleurisy the diet should be as dry as possible but should fully meet nutritional demands. It is wise to give as little liquid as possible; milk, water, and soups should be sparingly used; meat, eggs, cereals, and bread, with a minimum amount of milk, furnish a nutritious diet, and one that is believed to promote the absorption of the serum in the pleural cavity. Saline laxatives are advisable and diuretics, such as acetate of potash and diuretin, may be given in doses suited to the age of the child; these remedies, however, apply to the child and not to the infant. In the infant the disease is commonly purulent, and, even when it is not, the remedies above mentioned can do no good. A cardiac tonic, such as tincture of strophanthus or tincture of digitalis, should be given in every case. In empyema it serves as a supporting measure to the heart, and in serous pleurisy, by improving the circulation and acting as a diuretic, it promotes the absorption of the fluid. Whiskey should be given as long as the septic temperature continues. Sodium salicylate (from oil of wintergreen), or aspirin, in doses suited to the age of the child, are valuable in serous pleurisy but not in empyema. The salicylates are especially indicated in children of gouty or rheumatic parentage.

Paregoric, codein, or some other preparation of opium may be necessary to relieve the pain in the side, although it is advisable to avoid their use as long as possible; they are perhaps never indicated in the treatment of this disease in infants under eighteen months of age. In older children, however, when judiciously used, they may be of value in relieving the pain in the side or the irritating and paroxysmal cough which prevents sleep and increases nervous irritation. The sharp pain, which is aggravated by the cough and by respiratory movements, may sometimes be greatly modified by strapping the chest wall with adhesive plaster, or, as Jacobi recommends, by fastening a tight towel bandage around the entire chest. Counter-irritation with mustard paste or mustard plasters is also very generally recommended for the relief of pain. With the onset of the disease cold applications in the form of an ice-bag wrapped

in a towel may be applied to the affected side; later, hot fomentations or hot poultices give more relief.

Certain medicines, given by inunction, such as guaiacol one drachm, to one ounce of anhydrous lanolin, may be used to advantage. This ointment is to be thoroughly rubbed in, and then a warm poultice applied; it is of special value in tuberculous cases. Iodin and salicylic acid may also be given by inunction, and are to be used in the strength of one drachm to the ounce of anhydrous lanolin. These ointments are readily absorbed and pass directly into the lymph and blood channels; this is the only manner in which these drugs should be given to children under three years of age, since, when given in this way, they are more effective and do not disturb the gastrointestinal organs.

Aspiration of the pleural cavity for the removal of the fluid in serous pleurisy is not only a curative but at times it may be even a life-saving measure. It is perhaps not advisable to resort to aspiration in every case of serous pleurisy. In a minority of these cases the fluid will be spontaneously absorbed and a satisfactory convalescence established within a period of three weeks. But, on the other hand, it should be remembered that aspiration can do no harm, promotes convalescence, and diminishes the number of permanent adhesions. Aspiration is demanded when the fluid has accumulated in sufficient quantity to displace the heart or embarrass its action; when the pleural cavity is well filled and respiration is embarrassed, and in those cases in which the fluid does not commence to diminish in quantity during the second week of the disease. The increase or diminution in the quantity of fluid may be determined by the physical signs previously mentioned and by careful measurements of the affected side. If the tape measure shows the chest to be increasing in size the fluid is on the increase; if these measurements are found to be decreasing the fluid is gradually disappearing. In aspirating the pleural cavity, the Potain, or some other equally good aspirator, is to be used, and the aspirating needle must be large enough to allow a free flow of pus through it. In introducing the needle the same antiseptic precautions are to be observed as have been described above for the exploratory puncture. A sterile needle is to be introduced through the thoroughly cleansed skin, at a point where the exploratory puncture has located the serous exudate. It is well to introduce the needle just above the lower line of absolute flatness, so as to tap the lower portion of the fluid-filled sac. A sufficient quantity of serum is slowly withdrawn to relieve the pressure on the heart and lung. It is not necessary, neither is it wise, to withdraw all of the fluid; if the greater part is removed the remainder will probably be absorbed. In some instances it may be necessary to aspirate a second time. If, during the removal of the fluid, the patient complains of being faint, if the heart action becomes very weak, if a violent attack of coughing occurs, or if other uncomfortable symptoms supervene, the needle is to be immediately withdrawn.

Surgical Treatment of Empyema.—When pus is present in the

pleural cavity it must be evacuated at once by an incision between the ribs or preferably by a more radical surgical operation which comprehends the resection of a small portion of one or more ribs. To attempt to treat these cases by aspiration is, in most instances, a dangerous waste of time, yet Murphy advocates that the pus should be withdrawn from the abscess cavity by the introduction of a needle, and, following this operation, 60 c. c. of a 2-per-cent. solution of formalin in glycerin be injected into the cavity. He emphasizes the point that this solution should be at least twenty-four hours old. This process is to be repeated every two to four weeks until the fluid drawn off becomes serosanguinolent and sterile; it will then be absorbed. In very young children an intercostal incision into the abscess cavity, of sufficient size to permit the insertion of a drainage tube, is commonly successful. It should be made under proper antiseptic precautions, as low down over the pus pocket as possible so as to facilitate proper drainage. This simple operation may, as a rule, be made under a local anesthetic, such as a weak solution of cocain, injected into the superficial layers of the skin at the site of the operation. In introducing the knife, it is important to avoid the intercostal artery which lies along the lower border of the rib. Following the incision, one or two short drainage tubes, held by safety-pins, should be inserted into the cavity, and drainage may be facilitated by one of the suction methods later referred to, or the site of the operation may be well padded with gauze to absorb the pus and a light bandage applied to hold it in position. If the gauze method of dressing is used it should be removed one or more times daily. If the drainage is good a satisfactory recovery commonly results. This is especially true of the pneumococcic form of empyema, so common in young children. In children over three years of age, as well as in younger children, in whom the above simple operation is not successful, the abscess cavity should be drained by the excision of a small section of one or more ribs. In this operation the periosteum should be stripped back and the incision made through its posterior layer. In aggravated cases it may be necessary to make a rib exsection at two points of the chest, anteriorly and posteriorly, in order to obtain more perfect drainage. The opening or openings thus made should be large enough to receive two rubber drainage tubes, and through these the cavity may be irrigated with ordinary antiseptic solutions (not peroxid of hydrogen). The operation of preference, however, in practically all cases is to make one rib exsection rather low down over the pus pocket, insert two drainage tubes, and employ light continuous suction by one of the recently devised methods for this purpose. In this way the pus cavity is not only properly drained but the lung is kept in a state of expansion and sinuses are obliterated. Of the various types of apparatus devised for draining the abscess cavity and preventing lung collapse the following are the simplest and most generally applicable: Bryant's method, in which a deflated Politzer bag is attached to the drainage tube and strapped to the chest. Brewer's method, especially applicable in young children, consisting of a glass

funnel, securely placed against the chest with its wide mouth covering the drainage tubes, the suction being secured by means of a pump connected by a rubber tube to the small end of the funnel.

Collapse of the lung during operation may be prevented by Meltzer's method, modified by Elsberg. This consists in pumping air into the lungs during the operation for empyema through a tube, considerably smaller than the lumen of the trachea, passed far into the trachea through the glottis. Under this method the air enters the lung through the tube and escapes around the tube, and sufficient pressure is thereby maintained to prevent collapse of the lungs. A number of other methods have been devised having the same object as the Meltzer method. The ideal operation for acute empyema would be to evacuate the pus by thoracotomy, under a method which would hold the lung close to the chest wall, and then apply an air-tight dressing or, as Ransohoff suggests, grasping the lung and sewing it to the wound margin while under the differential pressure,

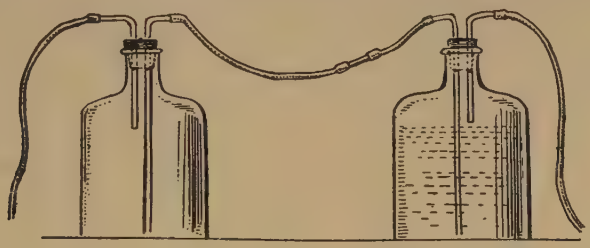


Fig. 78.—James Apparatus for Expanding the Lung.

and then applying one of the suction or ordinary drainage methods above described.

Following the operation the lung should be kept as quiet as possible for four or five days and then should be encouraged to expand by active breathing exercises, or by the use of the apparatus devised by James for expanding the lung. This consists of two bottles connected by rubber tubing, each being one-half filled with fluid. The child amuses itself by blowing the fluid from one bottle to the other and in this way obtains an excellent pulmonary exercise which assists in the expansion of the contracted lung.

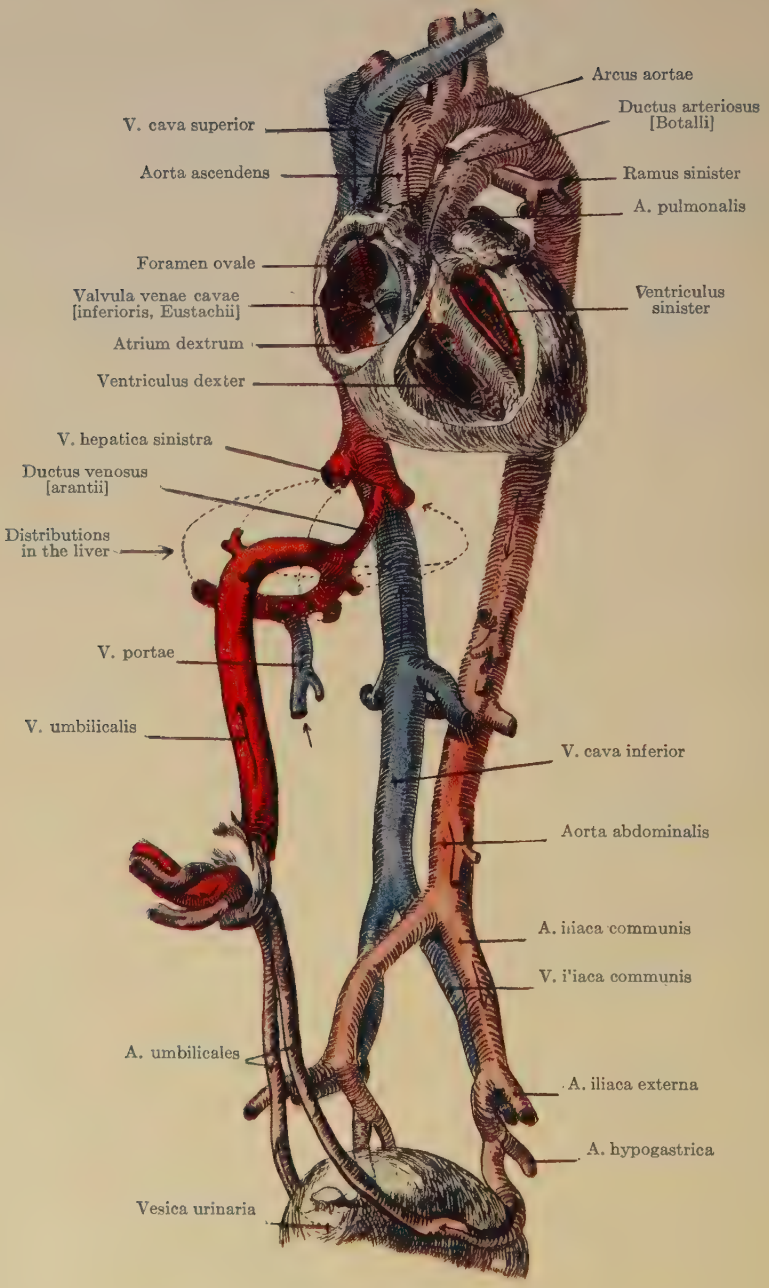
*Bilateral Empyema.*—This is comparatively rare, but when it does occur the left side should be operated upon first and the opposite side a week or two later. When rib excision is performed on the left side the right side may be aspirated, and, if necessary, this aspiration may be repeated from time to time to prevent the accumulation of pus in quantities sufficient to seriously impede respiratory movements.

*Chronic Empyema.*—Chronic empyema which has failed to respond to surgical methods combined with the fresh-air treatment, may be greatly benefited, and, in some instances, convalescence may be established by the use of autogenous vaccines as described in the chapter on Therapeutics of Infancy and Childhood. In obstinate cases it may be necessary to per-

form decortication of the lung, as recommended by Fowler, removing *en masse* the thickened visceral pleura and thus allowing the lung to expand.

Treatment During Convalescence.—Following the disappearance of all symptoms in empyema all forms of exercise involving the arms and chest must be carefully avoided. The child should live and sleep in the open air and should be given a carefully selected diet within the range of its digestive capacity and tonics containing malt, iron, arsenic, or cod-liver oil may be indicated. After a period of six or more months, when apparently all danger of a return of empyema has disappeared, the patient should be referred to an orthopedic surgeon for the correction of spinal and chest deformities. Properly directed breathing and gymnastic exercises may markedly diminish the resultant permanent deformity.

PLATE V.




SCHEME OF THE CIRCULATION OF THE BLOOD IN THE FETUS
(after Spalteholz).

## SECTION VIII

### *THE HEART*

The heart muscle of the child is strong, elastic, and bears strain with comparatively little injury, not only because of the very great elasticity of the muscle cells at this age, but also because the elastic tissue surrounding these cells is fully developed at the age of seven (Fahr). It is in a state of growth and functional development, and therefore readily undergoes hypertrophy when called upon to do more than the normal amount of physiological work over a long period of time.

The strength, elasticity, and healthful condition of the arteries during childhood greatly increase their efficiency in promoting and equalizing the circulation, and also minimize the ill effects on the circulation which result from inflammatory diseases of the heart and strain of the heart muscle during this period of life.

In early infancy the size of the heart and the capacity of the arteries are relatively larger than at any other period and the capillary circulation is much more active. The relatively large size of the great blood vessels, and of the openings through which they enter and leave the heart, is of special physiological and pathological importance, as they give an enormous advantage to the infantile heart in promoting circulation. The infantile heart acts rapidly (110 to 120 per minute), and drives a comparatively large blood stream, with little resistance, through large openings into large arteries which, by reason of their great elasticity, promote the rapid circulation of the blood so characteristic of infancy. These conditions account for the low blood pressure (80 to 90 m. m.). As the child grows older the heart increases in size and strength, but there is little or no change for five or six years in the size of the ostia, and thereafter the increase in the size of the heart continues to be very much greater than the increase in size of the ostia, so that the difference between the capacity of the heart and the capacity of the arteries gradually increases throughout childhood, and with this change there is an increase in blood pressure reaching 110 mm. at ten years of age. The total body weight of the adult is nineteen times that of the newly-born infant, but the heart is only fifteen times as heavy as at birth, so that the relation of heart weight to body weight is never again as favorable as in earliest infancy. (Hochsinger.)

The younger the child the more rapid and unstable is the pulse. During the first year the normal average varies from 100 to 140, and thereafter diminishes four or five beats a year, until at ten the average pulse is about 80. The arrhythmia so frequently observed in infancy is largely due to lack of inhibition, which results in an instability of the nervous mechanism of the heart; it is therefore of little pathological importance. Apart from the normal slight irregularity of rhythm of the infantile heart, which is most common during sleep, it is very easily influenced in its rate and rhythm by pathological conditions of all kinds. The rapid, irregular pulse, which is produced so readily from slight causes in infancy and early childhood, is not of such pathological significance as it is in later childhood and adult life. This is especially true of the rapid heart; the normally rapid pulse of the child may become almost uncountable from slight and evanescent causes.

During the first five years of life the heart increases in size, strength, and weight, but not in circumference (Beneke). From this time to puberty, while its openings from and into the great vessels increase very slowly in size, there is much greater increase in the size of the heart, and yet it does not keep pace in development with the chest cavity. The long time that the ostia remain almost stationary favors the muscular power of the heart. The apex beat in infancy is commonly in the fourth intercostal space, in early childhood in the fifth interspace, and in later childhood slightly lower. In infancy it may be just outside and in early childhood just inside the mammary line; in the older child it is found well within this line. This change in location of the apex is due not alone to the growth of the chest cavity, but is also due to the swinging of the heart downward and inward from the slightly oblique position it occupies in infancy to its perpendicular position in childhood. The area of cardiac dullness, while it actually slowly increases with the growth of the heart, gradually continues throughout childhood to occupy a smaller proportion of the chest cavity, and the external border of this dullness is thereby slowly moved from just without to within the mammary line. Its outer border in infancy is slightly outside the mammary line, its inner border the midsternal line, its lower margin the fourth or fifth interspace, and its upper margin the second interspace. Enlargement of the heart is determined by a displacement of the apex beat downward or outward, and by an increase in the width of the cardiac dullness extending either 2 or 3 c.m. beyond the midsternal or the mammary lines.

## CHAPTER LIV

### CONGENITAL HEART DISEASE

**Etiology.**—The character of the fetal circulation and the changes which occur in it at birth are necessary to the understanding of the etiology of

congenital heart disease. The most important peculiarities of the fetal heart are the direct communication between the two auricles through the foramen ovale, and the large size of the Eustachian valve which serves to direct the blood entering the right auricle from the inferior vena cava directly through the foramen ovale into the right auricle. The important peculiarity in the arterial system of the fetus having a bearing on congenital heart disease is the communication between the pulmonary artery and the descending aorta by means of the ductus arteriosus.

At birth the foramen ovale is normally closed by the increased pressure in the left auricle and thereafter there should be no direct interchange of blood between the auricles. The function of the ductus arteriosus should also cease at this time and thereafter there should be no direct communication between the pulmonary artery and the descending aorta.

Malformations, due to arrested or perverted development, are the most common causes of congenital heart disease. They are frequently associated with the persistence of the above-named fetal conditions, namely a patulous foramen ovale and ductus arteriosus, and they are also commonly associated with congenital deformities elsewhere in the body, showing that they are due to the same general causes, producing perverted development in various parts of the body. Consanguinity and neurotic disease in the parents, and early psychic influences acting on the pregnant mother, may be etiologically related to these cases. Heredity may also be a factor. Fetal endocarditis is also an important cause of congenital heart disease. The acute infections, rheumatism, syphilis, tuberculosis, and traumatic lesions are mentioned as possible factors of this condition.

**Symptomatology.**—General Symptoms.—*Cyanosis,* which is one of the most characteristic symptoms, is due to a venous condition of the blood and not to passive congestion of the skin from weak cardiac action. In aggravated cases, however, the cyanosis is increased by physical exertion and the superficial veins of the skin may gradually become more or less chronically congested. Cyanosis, while a common symptom, does not occur in all forms of congenital heart disease. It is nearly always present in that most frequent of all lesions, congenital stenosis of the pulmonary artery, and always occurs in a very pronounced form in the rare congenital condition where the aorta takes its origin from the right heart. It is commonly absent, however, in lesions of the ventricular septum and in open ductus arteriosus. It may occur during the first days of life, or it may not appear until the infant is two or three years of age. The earlier and deeper the cyanosis the graver the prognosis. In the less severe cases cyanosis may come and go, and be greatly aggravated by excitement and exertion.

*Bulging of the precordia* is an early and frequent symptom. A horizontal increase of precordial dullness, murmurs, and retinal changes may also be noted.

*Dyspnea,* which is a marked symptom of congenital heart disease, is,

as a rule, in direct proportion to the severity of the cyanosis. It is aggravated by mental excitement and physical fatigue. Clubbed fingers are found, especially in those cases in which there are cyanosis and dyspnea. The terminal phalanges are knob-shaped and have a bluish tinge. The toes may also show this same deformity.

The *blood picture* associated with the cyanosis presents the following characteristics. The blood has a dark blue color, due to excess of $CO_2$ and deficiency of O. The red blood corpuscles are increased in size (macrocythemia). The amount of hemoglobin in each corpuscle is increased and the great increase in the percentage of hemoglobin gives to the blood a very high specific gravity, sometimes reaching 1,070. The most characteristic blood change, however, is polycythemia; the number of red cells may reach 7,000,000, or even more. This increase goes hand in hand with the cyanosis, a high red-cell count being associated with the worst cases of cyanosis. The above blood changes are not found in congenital heart diseases unassociated with cyanosis.

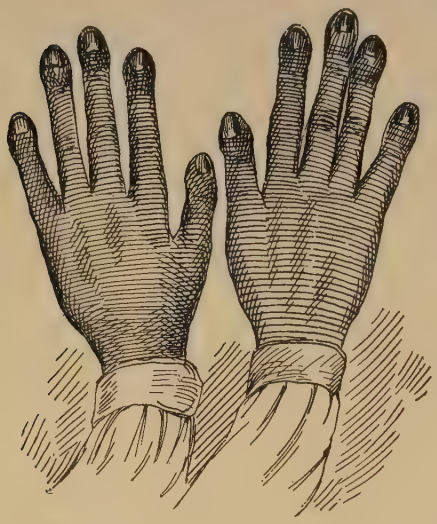


FIG. 79.—CLUBBING OF THE FINGERS IN CONGENITAL HEART DISEASE.

The *malnutrition* and *defective development* will be in direct proportion to the physiological incompetency of the heart. Curvature of the spine and other rachitic deformities are common.

*Enlargement of the heart* from hypertrophy or dilatation is not so marked or characteristic in the common forms of congenital heart disease as in acquired heart lesions, but when it does occur it extends to the right beyond the sternum. In some of the rarer congenital conditions (patulous ductus arteriosus) the cardiac hypertrophy may be very great.

Systolic *murmurs* are by far the most common. They may be diffused over the whole cardiac area, but they are commonly heard more distinctly at the base of the heart; diastolic murmurs are very rarely congenital. Hochsinger says: "Abnormally loud cardiac murmurs in infants and little children are an almost infallible sign of the congenital nature of the existing heart affection."

SPECIFIC LESIONS.—The differential diagnosis of the various lesions occurring in congenital heart disease is, as a rule, difficult and ofttimes impossible. The chief reason for this difficulty lies in the fact that these lesions rarely occur singly; in nearly all instances one or more congenital defects are associated. Of the various lesions of congenital heart disease two are of special interest to the clinician, namely stenosis

of the pulmonary artery and congenital defects of the ventricular septum. These two deformities are of special interest because they make up the great majority of the cases and because they are frequently associated in the same case. In addition to these, the following deformities occur: open ductus arteriosus, open foramen ovale, aortic stenosis, abnormalities in the origin of the great vessels and valvular anomalies involving any of the valves of the heart. These latter anomalies, however, are comparatively rare, and when they occur are usually associated with either pulmonic stenosis or defects in either the ventricular or auricular septum.

*Stenosis of the Pulmonary Artery.*—Most of these cases are due to developmental defects; the remainder to fetal endocarditis. This is the most common form of congenital cardiac defect; 68 per cent. are due to this cause (Peacock and Keith). It ranks first in clinical importance, because of its rather clear symptom-complex and because these cases may live for a long time and require medical supervision.

Early cyanosis, associated with dyspnea, clubbed fingers, and the characteristic blood picture previously noted under General Symptoms, is an important part of the symptom-complex of this condition. In the great majority of cases there is a loud, rough, long systolic murmur heard at the base, its maximum intensity being in the second intercostal space just to the left of the sternum; it is not transmitted to the arteries of the neck. With this murmur a distinct thrill may be felt by placing the hand over the cardiac area; the absence of this sign may depend upon complicating cardiac defects. The heart is enlarged; the cardiac dullness extends to the right. The second pulmonary sound is not accentuated and may be absent. The above signs and symptoms, when they exist, are pathognomonic of pulmonary stenosis, and in the great majority of these cases the symptom group is sufficiently complete to make the diagnosis clear. In a few instances, however, either by reason of associated defects or from inexplicable causes, this symptom group is so modified that the diagnosis cannot be definitely made.

Prognosis.—Many of these cases live to adult life. Those that are complicated with severe septum defects and other anomalies die early from tuberculosis, acute endocarditis, and other causes. Deep cyanosis, and continuous polycythemia indicate an early termination of the disease.

*Defective Interventricular Septum.*—This is one of the most common of congenital heart lesions. It may be the only malformation, but, as a rule, it is associated with pulmonary stenosis, which greatly complicates the symptom-complex. It is also usually associated with an open foramen ovale, which condition, however, adds little to the symptom group. In rare instances the entire septum may be absent, but partial defects, usually located at the base, make up the majority of these cases. Of the various cardiac anomalies this is the one most commonly associated with deformities in other parts of the body.

When this cardiac defect exists as an independent condition it is characterized by a long, harsh, systolic murmur heard over the whole car-

diac area, having, as a rule, its point of greatest intensity, according to Roger, in the upper third of the precordial region. It is not heard over the great vessels of the neck, but is transmitted downward. A distinct cardiac thrill may be felt in many of these cases and a marked systolic retraction over the precordium and epigastrium may be seen. Cyanosis and its accompanying symptoms are, as a rule, absent; when present they are usually slight or intermittent. It is a notable fact that the signs and symptoms of this condition may be altogether out of proportion to the extent of the lesion. In some instances extensive defects in the interventricular septum exist without symptoms, the condition being discovered post mortem. In other instances small defects give rise to loud murmurs. The frequent association of this cardiac anomaly with pulmonary stenosis leads to a confusion of the two symptom groups. In such instances cyanosis and its accompanying symptoms are an important part of the combined symptom-complex. Hochsinger says that in septum defects the systolic murmur is associated with an accentuation of the second sound at the pulmonary area, and that this materially assists in differentiating these murmurs from those produced by pulmonary stenosis.

Prognosis.—In uncomplicated cases the patients may live to adult life; the majority of them die during childhood.

*Persistent Patulous Ductus Arteriosus Botalli.*—This, as a clinical entity, is a rare condition, but occurs more frequently in association with pulmonary stenosis and defective interventricular septum. Cyanosis is, as a rule, absent in these cases, and the skin may even present a pallid wax-like appearance. The dilated pulmonary artery running across the base of the heart presents a ribbon-like band of dullness in the first and second intercostal spaces (Gerhardt). In Röntgen ray pictures this artery produces a shadow "like a cap" covering the general cardiac shadow (Arnheim); this is a most important point in differential diagnosis. There is a loud, long, buzzing, systolic murmur most distinct at the base and transmitted not downward but into the carotids, especially the left. The second pulmonary sound is accentuated, a distinct systolic thrill may be felt, and, with a systolic, a diastolic murmur is often heard following the accentuated second pulmonic sound. Hypertrophy of the right and sometimes of the left ventricle may produce a great increase in the area of cardiac dullness, and the systolic pulsation described by Gerhardt may be seen in the second left interspace where the heart strikes the chest wall. The above clinical picture is greatly complicated when the patulous ductus is associated with pulmonary stenosis, as the blending of the two syndromes may produce a symptom-complex in which it is difficult to make out with clearness either condition.

**Treatment.**—The treatment of congenital heart disease is purely symptomatic and quite unsatisfactory, since all that one can do is to promote the comfort and prolong the lives of these children. Attacks of cyanosis may be relieved by the administration of oxygen. During the first days and months of the life of the child it may be necessary to keep up the

body temperature by artificial heat. These feeble infants are especially prone to gastrointestinal disturbances and must therefore be fed with great care; breast-milk is ofttimes the only food upon which they can thrive. If the child lives, care must be exercised to prevent the development of spinal curvature which frequently occurs. As children of this type always remain weak and undeveloped they must throughout their lives be carefully protected from contagious diseases.

Congenital heart lesions require digitalis only when the symptoms of myocardial insufficiency are very marked.

## CHAPTER LV

### ACUTE ENDOCARDITIS

Acute endocarditis is an inflammation of the endocardium, most marked on and near the valves. In children especially the whole heart muscle is more or less involved. It is essentially an infection, but the disease varies greatly in severity from the simple cases which run a benign course of two or three weeks' duration to the septic or so-called ulcerative cases, which, with few exceptions, terminate fatally. The severity of these cases depends partly upon the susceptibility of the individual, but more on the character of the microorganism which is producing the inflammation. In fetal life the right side of the heart is usually affected. After birth the left is chiefly involved.

**Etiology.**—It is extremely rare in infancy, uncommon before the fourth year, and thereafter increases in frequency until the tenth year; between this age and the fifteenth year of life it is most commonly seen. It is observed most frequently in the late winter and spring. Heredity is an important predisposing factor.

Rheumatism is the great exciting cause. Rheumatic arthritis or chorea is associated with endocarditis in 70 to 80 per cent. of the cases; in order to understand this relationship, it should be remembered that rheumatism is a general febrile disease of infective origin, whose chief manifestations, as Cheadle has taught, are non-suppurative polyarthritis, acute inflammatory diseases of the heart, and chorea. One or all of these manifestations may be present at the same time, or any one may take precedence in the order of their development, but most commonly the arthritis precedes the heart disease and the chorea. Polyarthritis and chorea are therefore rheumatic syndromes, commonly associated with endocarditis, and their presence should make the physician ever watchful for the development of heart symptoms, marking the insidious onset of inflammatory disease of this organ. On the other hand, in searching for evidence of rheumatism to explain an existing endocarditis, the physician should keep in mind the mild character of the rheumatic polyarthritis that occurs in childhood. In many instances there will be a history of a mild febrile attack, with per-

haps slight indefinite pains, and joint tenderness so mild as to be discovered only on pressure.

The lymphoid ring of the pharynx is the common portal of entrance for bacteria (Jacobi), and Packard has called attention to the fact that endocarditis in children is very commonly preceded by tonsillitis. Influenza, scarlet fever, tuberculosis, pneumonia, and septic processes in general may be complicated or followed by an endocarditis.

**Pathology.**—The pathological anatomy of endocarditis in childhood is very similar to that which occurs in the adult. The different forms of this disease have not been definitely associated with specific microorganisms; streptococci and staphylococci are most commonly found; the pneumococcus, gonococcus, typhoid bacillus, and other microorganisms are more rarely observed. The valves are the sites of the most marked lesions, the mitral being by far the most commonly affected. The endocardium is thickened, its superficial epithelium is destroyed, and fibrous vegetations occur, which thicken and prevent the proper closing of the valves. Small particles may be separated from these vegetations and carried by the blood to distant organs, producing infarcts and secondary infections. As the inflammation subsides these fibrous deposits may be in great part absorbed, but resulting contractions of the cordæ tendinæ or deformities of the valves themselves commonly result in their incomplete closure with a resultant incompetency or leakage of the valve. In the ulcerative form the vegetations are broken down by ulcerative processes and septic particles are cast into the blood stream, thus producing a general septicopyemia with localized abscesses in various organs of the body.

**Symptomatology.**—An insidious onset is characteristic of endocarditis in childhood. This disease usually develops with few or no symptoms directing attention to the heart itself. Fever is present, but the irregular temperature curve is usually mistaken for an exacerbation of the fever of the rheumatism or other acute infection with which the endocarditis is commonly associated. Broadbent says that an intermittent or irregular fever in childhood, which resists quinin, often indicates endocarditis. Epistaxis may occur. Shortness of breath, rapid and irregular action of the heart, with slight precordial distress, may be present in some cases; this is especially true where the myocardium is involved and cardiac dilatation is the first evidence of endocarditis. In other instances progressive wasting and anemia, unaccompanied by acute symptoms, may call for a careful physical examination, which reveals the cardiac bruit of endocarditis. The insidious onset of this condition, therefore, calls upon the physician to make a careful physical examination of the heart daily in all cases of rheumatism and other infections which may be etiologically related to endocarditis.

The most important clinical sign of this disease is a *low, blowing systolic bruit, heard most distinctly at the apex, and transmitted toward the axillary region;* this murmur means mitral regurgitation. As the mitral valve is almost exclusively affected in this disease the heart murmur

has its point of greatest intensity almost always at the apex. In a small minority of these cases there is a mitral stenosis, which produces a presystolic apical bruit, usually associated with, but rarely independent of, the systolic apical bruit. Both systolic and diastolic apical bruits may exist for a considerable length of time without producing reduplication of the second sound over the pulmonary valve. Aortic disease is infrequent in the acute endocarditis of children. In this condition systolic murmurs are the more common, and have their point of greatest intensity in the second intercostal space to the right of the sternum; they are due to the roughness of the aortic valves and ostium. In rare instances diastolic aortic murmurs may be heard in the same location. Percussion is of little value, since there is little increase in the size of the heart during an acute attack, except in cases of acute dilatation, and here the symptoms of cardiac distress are so marked that the nature of the lesion can scarcely be overlooked.

**Septic Endocarditis.**—Septic endocarditis is a term now in general use to describe those cases of endocarditis which are complicated by a general septicopyemia. They do not represent a separate or distinct disease, but are produced by the same microorganisms sometimes found in simple endocarditis. In simple endocarditis these microorganisms confine their ravages almost exclusively to the heart; occasionally they are transmitted through the blood stream to distant parts of the body, where they may produce inflammatory infarcts. In the septic form, however, there is a bacteriemia of the same microorganisms which have produced the endocarditis. The focus for the distribution of this general infection is in the ulcerated heart valves, from which the septic microorganisms are thrown into the blood stream and are generally distributed throughout the body, producing septic foci in distant organs; in this way the general clinical picture of septicopyemia is added to that of the existing endocarditis. These cases are not necessarily septic or ulcerative from the start. Some years ago I reported three fatal cases of septic endocarditis, following ulcerative tonsillitis. In each instance the patient recovered from the first attack of endocarditis with damaged mitral valves, and months later secondary attacks of ulcerative tonsillitis were followed by endocarditis, which assumed the septic or ulcerative type and ended fatally. In one of these cases the necropsy showed ulceration of the mitral valve on both leaflets.

The term malignant, used to describe these cases, is a misnomer, which has added confusion to this subject, as they are not malignant nor are they necessarily hopeless. Adams reports three recoveries in forty-seven cases collected from the literature (one of these was his own case), and the probabilities are that these figures do not represent the full percentage of recoveries. The important point to bear in mind is that the majority of these cases are not septic in the first attack and that subsequent attacks are produced by a secondary invasion from the same microorganisms which produced the first attack, but which, in the meantime, have been

held quiescent in foci commonly located in the tonsils, but sometimes in the lungs, pelvis, and subcutaneous tissues. In any one of these attacks an ulcerative endocarditis may develop which may result in a general septicopyemia.

*Symptomatology.*—The symptoms of septic endocarditis are those of severe endocarditis plus a general septicopyema. One of the most important diagnostic signs is the finding of septic organisms in the blood. These cases are characterized by high and variable temperatures, such as are seen in sepsis, the temperature commonly approaching or falling below the normal and rising to 104°, 105°, or 106° F. within the next twelve hours. There are great prostration, delirium, chilly sensations, and the body is frequently covered with a petechial rash. A high leukocyte count is present. These cases usually end fatally, but, under modern methods of treatment, the death rate promises to be less than it has formerly been.

**Diagnosis.**—Accidental murmurs due to anemia, cardiac neuroses, and other causes may occur and are to be differentiated from the bruits produced by endocarditis. The above-mentioned murmurs are commonly heard most distinctly at the base, are not transmitted to the axillary region, and occur in non-febrile conditions. A relative insufficiency of the mitral valve may be caused by cardiac dilatation; the murmur thus produced is associated with enlargement of the heart and displacement of the apex beat, and appears, as a rule, rather suddenly under conditions producing heart strain. The pericardial friction murmur rarely causes confusion; it is not transmitted, is intermittent in character, and its point of intensity is usually located at the base.

**Prognosis.**—The prognosis in acute simple endocarditis, so far as life is concerned, is good. The great majority of these cases recover in three or four weeks. In a small minority there is complete recovery, but in the great majority of instances incompetency of the mitral valve occurs, which results in a leakage at the valve and a crippling of the heart, which is compensated for by cardiac hypertrophy. Secondary attacks of endocarditis may result in further crippling of the heart and in the production of chronic valvular disease. In rare instances an ulcerative endocarditis develops, associated with a septicopyemia; the prognosis in these cases is very bad, the great majority of them terminating fatally.

**Prophylaxis.**—This comprehends the careful treatment of rheumatism and of all the acute infections. According to Forchheimer, the alkaline treatment of rheumatism diminishes the tendency to fibrin formation and thereby to endocarditis. In all of the acute infections, especially rheumatism, the heart must be carefully watched and the patient confined to bed during the acute stages of these diseases, and, in the event that symptoms develop referable to the heart, a longer period of absolute rest in bed must be insisted upon. The most important prophylactic treatment, however, consists in the prevention of second and third attacks of endocarditis. If the primary attack was associated with chorea, rheumatic arthritis, or

other symptoms indicating the rheumatic origin of the disease, then the subsequent life of the patient, at least for a number of years, should be carefully regulated to prevent second attacks of rheumatism, and he should take at intervals, especially during the winter months, courses of medical treatment which include the alkalies and salicylates. These medicines materially assist in warding off the second and third attacks. These patients should spend their winters for a number of years in mild climates; where this is impossible they should be protected by woolen underclothing and proper footwear from the cold, damp weather of the winter months. But the most important prophylactic measures in these cases are the removal of diseased tonsils and adenoids, and the daily disinfection of the throat and nose during the winter months with mild alkaline antiseptics. As previously noted, the lymphoid ring of the pharynx, which includes the tonsils and adenoids, is not only the common portal of entrance for the germs which produce endocarditis, but they offer a hiding place for them in the intervals between the attacks, which makes it possible for slight causes, such as exposure to damp cold, to set up a tonsillitis, to be followed by another attack of endocarditis. It is my belief that if the tonsils and adenoids in all these cases were removed following the first attack of endocarditis, and thereafter during the winter months the throat was daily douched with an alkaline antiseptic, relapsing endocarditis would be less common, and septic endocarditis could, in most instances, be prevented.

**Treatment.**—The all-important part of the treatment is prolonged rest in bed, and unless this is carried out satisfactorily other curative measures are without avail. This implies that the child must be kept as quiet as possible, not allowed to sit up, and not permitted to get out of bed for any purpose whatever. The younger the child the more difficult it is to carry this out in a satisfactory manner. During the early acute inflammatory stage an ice-bag or coils of cool running water should be applied over the heart; this is especially indicated when precordial pain and rapid heart action are present. In the less severe cases this application should be made at intervals during the day and under no conditions should it be used when it interferes with normal sleep, as sleep is almost, if not quite, as important as rest in bed. The surroundings should be as quiet as possible so as to avoid mental stimulation and nervous excitement. If the child be restless, nervous, and sleepless the bromides are indicated, and occasionally opiates may be necessary to secure proper rest; where these are indicated, a hypodermic injection of 1/20 to 1/50 of a grain of morphin may be given at bedtime. All sedative medication, however, should be discontinued as soon as possible and should be given only in those cases where the nervous irritation is markedly interfering with the rest of the child. The diet should be carefully selected to suit the wants of the individual child; milk, cereals, and eggs may form the basis of this diet during the acute stage. The bowels should be kept open by mild cathartic medication. If rheumatism be present, the salicylates should

be given until these symptoms are under control; aspirin, salol, and wintergreen sodium salicylate may be used, as recommended in the chapter on Rheumatism. When other internal medication is not indicated, bicarbonate of soda or some other alkali may be the routine treatment; in older children two or three drops of tincture of nux vomica may be given with each dose of the alkali. In the treatment it should be remembered that all medicines that upset the stomach or interfere with the appetite do more harm than good. Where the heart is weak and the pulse irregular tincture of digitalis or strophanthus may be used in from 3 to 5-drop doses. In most cases of primary simple acute endocarditis cardiac stimulants, however, are not only unnecessary but are usually contraindicated.

The important question to decide in every case is the length of time the patient should remain in bed; even in the mild cases one month is the minimum time. When the physician has decided that the patient has recovered sufficiently he may test the action of the heart by allowing him to sit up in bed, and, if no ill effects follow, within a few days he may be placed in a chair for a few hours during the day, and later may be allowed to walk across the room. In this way the patient, during convalescence, should be carefully guarded against overexertion until the heart is able to do the work ordinarily required of it. If the patient be guided in this way to a satisfactory recovery, and future attacks of endocarditis be prevented, the heart, in most instances, even though a loud mitral murmur persists, acquires a physiological competency which will enable the individual, throughout a long life, to follow many of the wage-earning vocations.

*Ulcerative Endocarditis.*—This is the same as that of ordinary endocarditis plus the treatment for septicopyemia. The antistreptococcic serum, and collargolum in the form of unguentum Credé should be used as outlined in the chapter on Scarlet Fever. A few cases have recovered under the use of these remedies, and, as they can do no harm, they should be given in every case. The treatment by homologous vaccines offers a chance for recovery, especially in the so-called Schottmueller's disease. From blood cultures the microorganism causing the trouble is isolated, and from this organism vaccines are made according to Wright's method. If these vaccines be administered early in the course of the disease good results may be hoped for in some cases.

## CHAPTER LVI

### MYOCARDITIS AND ACUTE CARDIAC DILATATION

### MYOCARDITIS

Myocarditis is very common in childhood. It is usually produced by bacterial toxins and is therefore a common complication of the acute infections, especially diphtheria, scarlet fever, influenza, pneumonia, typhoid

fever, and whooping-cough. The toxins act directly upon the cardiac nerves and muscles, producing parenchymatous degenerations. The muscular fibers may show granular, hyalin and fatty degeneration, and under this change the heart muscle becomes weak, flaccid, and readily undergoes dilatation.

Myocarditis may also be produced by the bacterial invasion of the organ along the line of the blood vessels and connective tissue; in this form true inflammatory changes, involving especially the interstitial tissue, are produced. The most common offending organisms are streptococci, staphylococci and pneumococci, and the heart muscle itself may be infiltrated with pus cells and may be the site of small abscesses. These cases commonly result from septic emboli carried to the heart, and may occur therefore as a complication of scarlet fever, diphtheria, ulcerative tonsillitis, septicopyemia, osteomyelitis, and other diseases characterized by sepsis. In other cases the myocarditis may occur as a complication of endocarditis and pericarditis; in many of these rheumatism is the exciting cause.

**Symptomatology.**—The PARENCHYMATOUS FORM of myocarditis, due to bacterial toxins, is of special interest to the physician because it is the most common form and the one in which his skill can be of the most value. Symptoms of parenchymatous myocarditis may develop at any time throughout the course of the above-named infections, but they occur more commonly during the stage of convalescence. An intermittent pulse and irregularity in the cardiac rhythm may be the first symptoms announcing the onset of myocarditis, and they are especially significant when they occur in diphtheria, scarlet fever, pneumonia, and severe forms of influenza. As the disease progresses apical systolic murmurs appear, the pulse becomes rapid, flickering and irregular, and the apex beat may be feeble and difficult to locate. As cardiac dilatation develops, the heart sounds may be indistinct, and other and more complicated cardiac murmurs may occur, and the patient commonly complains of pain in the precordial region and suffers from nervous unrest. Pallor, cold extremities, dyspnea, and cyanosis may be present. In diphtheria the above symptoms, when associated with syncope and vomiting, are especially ominous and not infrequently presage death from myocardial insufficiency. In the milder cases, however, a modification of the above symptom group may continue for days or weeks until final recovery is established. The irregular and intermittent pulse sometimes continues for months or is readily developed on slight muscular exercise.

INTERSTITIAL MYOCARDITIS presents the same symptom group as that above outlined for the parenchymatous form. The diagnosis therefore of this condition can only be inferred from the fact that it is preceded by or associated with endocarditis, pericarditis, or septic processes. In these latter cases a septic type of temperature is generally present.

**Prognosis.**—The prognosis in the parenchymatous form is, as a rule, good and recovery, when it occurs, is usually complete. In the inter-

stitial form the prognosis is bad. Most of the cases die, and in those that survive recovery is, as a rule, incomplete, the heart muscle being permanently injured and frequently embarrassed by a coexisting endocarditis or pericarditis.

## ACUTE CARDIAC DILATATION

Acute cardiac dilatation is commonly the result of a preceding myocarditis, but, on the other hand, when it does occur as a primary condition it is almost always followed by more or less myocarditis. The two conditions are therefore inseparably associated in the medical mind, but, notwithstanding this, acute cardiac dilatation deserves separate consideration.

**Etiology.**—Influenza and whooping-cough may cause this condition in infants and in young and delicate children. The dilatation of the heart in whooping-cough is largely produced by the strain on the cardiac muscle, which results from an overful heart attempting to force blood through the cardiac ostia under the greatly increased resistance which occurs during an acute paroxysm of this disease. I also believe with Forchheimer that the violent paroxysmal fits of coughing, ofttimes seen in influenza, may produce cardiac dilatation. The toxins of influenza may also weaken the heart muscle and thus predispose to dilatation. Acute dilatation may result in rapidly growing malnourished children, especially during the pubertic period, from the severe strain thrown upon the heart by bicycle riding, foot-racing, jumping-the-rope, and other forms of violent exercise. It is most commonly seen, however, as a condition secondary to acute myocarditis, and is especially to be feared and watched for in the parenchymatous form of this disease, which is produced by the toxins of the acute infections. It also occurs with the failing compensation of chronic valvular disease and may occur as one of the earlier symptom groups of acute endocarditis.

**Prognosis.**—The prognosis depends largely upon the cause; where mechanical conditions are wholly or in great part responsible for the dilatation recovery is, as a rule, rapid and complete. When acute toxic myocarditis is the cause the prognosis is grave, but many of these cases end in complete recovery. When occurring as a symptom group of acute endocarditis the prognosis, so far as the symptoms of dilatation are concerned, is on the whole favorable. When occurring as a symptom of failing compensation in chronic valvular disease the prognosis is grave, yet many of these cases have repeated attacks of acute dilatation from which they at least partially recover.

**Diagnosis.**—The diagnosis of this condition must be made in connection with the etiological factors which produce it. The physical signs in every instance are the same, but the general symptoms vary materially with the cause which has been operative in producing the dilatation. A rapid increase in the area of cardiac dullness with a marked displacement of the apex beat downwardly and outwardly always occurs. The cardiac dull-

ness may extend from outside the mammary line to the right of the sternum, and the apex, which is feeble and diffused, may be felt in the fifth or sixth interspace well outside the mammary line. A soft systolic murmur may sometimes be heard at the apex, and the second sound over the pulmonary area may be accentuated. This acute enlargement of the heart, made out by physical signs, occurring with any of the conditions above noted as being associated with the etiology of acute dilatation, may be accepted as proof of the existence of this condition.

Acute dilatation is not always manifested by the same symptom group. Its presence, however, may be suspected when etiological conditions favorable to its development are followed by sudden syncope, rapid breathing (tachypnea) and rapid and irregular heart action. In infants especially a tendency to somnolence follows this acute prostration. From these alarming symptoms the child may gradually recover and present for a time the milder symptoms of cardiac distress above outlined under Acute Myocarditis. Repeated attacks of this kind may occur, somewhat milder in character, as the disease progresses to a favorable termination, or death may result at any time from complete myocardial insufficiency. In older children cardiac dilatation may produce a symptom group closely resembling angina pectoris. These cases, when associated with acute or chronic endocarditis, may present the clinical picture of angina sine dolore described by Musser, the absence of pain being due to the relief which the mitral insufficiency gives to the interventricular tension. In one such patient, nine years of age, whom I observed, the respirations reached 97 per minute, were shallow in character, and not accompanied by cyanosis. During an attack she sat up in bed with body rigid, shoulders elevated, and head thrown back. Her eyes were fixed and staring; there was an expression of fear and anxiety on her face, but when asked if she had pain she answered "no." The symptoms in this case were associated with an acute mitral insufficiency due to endocarditis. The angina symptoms were readily controlled by nitroglycerin and the patient made a satisfactory recovery, leaving the hospital with full compensation for a well-marked mitral insufficiency.

## PROPHYLAXIS AND TREATMENT

**Prophylaxis.**—Rapidly growing anemic children should not be permitted to engage in forms of physical exercise which will overstrain the heart. This is especially important in children suffering from functional disturbances of this organ. In whooping-cough all violent exercise should be prohibited, and in those cases where the paroxysms are very severe and are associated with marked disturbance of the heart action it may be necessary to confine them to bed for a number of weeks until the dangers of acute dilatation and myocarditis are passed. In diphtheria, pneumonia, and influenza it is of the greatest importance that the heart should

be carefully watched, so that with the first symptoms of cardiac distress the patient may be treated for myocarditis.

**Treatment.**—This is much the same as in acute endocarditis. Absolute and prolonged rest should be insisted upon. The patient should not only be confined to bed but he should not be allowed to do anything for himself that can be done by others; a fatal issue in many cases has been precipitated by the effort of sitting up in bed. During early convalescence great care must be used in determining the amount of exercise the patient may take with safety. An icebag over the precordial region is indicated if there be cardiac pain and rapid heart action. Caffein sodium benzoate or salicylate (½ to 2 grains), digitalis (2 to 5 drops of the tincture), or strychnin (1/100 to 1/200 of a grain) may be given, as indicated, at intervals of three or four hours. Camphor, dissolved in sterile oil, may be given hypodermically. In most cases one depends almost entirely upon the cautious use of tincture of digitalis or tincture of strophanthus; these drugs may be given at four to six-hour intervals and in 2 to 5-minim doses, according to the age of the child. If the action of the heart is improved under their administration they should be cautiously continued. If the use of digitalis and strophanthus is not followed by good results caffein, strychnin, or camphor may be administered hypodermically. In some instances, where the nervous unrest is very marked, morphin, in 1/20 to 1/50-grain doses, given hypodermically, acts very kindly and may be repeated as indicated. Alcohol in the form of good whiskey or brandy is of value. During convalescence iron and arsenic may be indicated.

In the treatment of acute cardiac dilatation, the removal of four or five ounces of blood from the median cephalic vein may give great relief and tide the patient over the attack. The application of leeches over the region of the liver is also recommended. My own experience teaches me that nitroglycerin is preferable to bleeding; it is indicated when the heart is in severe distress and may be discontinued when the acute symptoms threatening cardiac paralysis have passed away.

## CHAPTER LVII

### CHRONIC VALVULAR DISEASE

Chronic valvular disease is almost always due to endocarditis. This applies to the congenital as well as the acquired form. Not infrequently, however, the primary attack of endocarditis is overlooked, and the disease first comes under the observation of the physician in its chronic form; this is especially true in hospital practice. In most of these cases there is a history of a previous attack of acute rheumatism, or of some other acute disease, which leads to the belief that acute endocarditis occurred during that attack.

The mitral valve is affected in about 90 per cent. of the cases, and in the great majority the lesion is that of mitral insufficiency. Mitral stenosis, however, may occur as an independent lesion, or insufficiency and stenosis of this valve may be associated. Aortic disease, as an independent lesion, is more common in boys than girls. It is rare in childhood; stenosis is more common than insufficiency. Arteriosclerosis and other causes of aortic disease in the adult are not present in the child. Aortic lesions, especially stenosis, occur more commonly with mitral disease than as independent affections. Chronic disease of the pulmonary valves is very rare and usually congenital.

Chronic endocarditis is rarely seen as early as the second or third year of life, but from this time on it occurs with increasing frequency throughout childhood. Following an acute attack of endocarditis, which has permanently damaged the valves, especially the mitral, the young and growing heart muscle of the child rapidly hypertrophies to the extent that the lesion is largely compensated. Under this compensation the heart is able to perform its ordinary physiological duties and the individual may live a useful and comparatively healthful life, provided the heart is not overstrained or subsequently damaged by a second or third attack of acute endocarditis.

**Mitral Regurgitation.**—This is the condition found in the vast majority of the cases of chronic valvular disease in childhood. The mitral valve is incompetent and with each systole of the ventricle a portion of the blood is forced back through the leaking valve into the left auricle; this results in dilatation of the auricle, and, to accomplish the increased work thrown upon them, both auricle and ventricle hypertrophy. If this results in complete compensation the circulation is maintained, the right side of the heart is protected, and all goes well, the child suffering comparatively little inconvenience from the lesion.

Symptomatology.—Compensation, almost if not quite complete, is the condition usually found in the chronic mitral insufficiency of childhood. In these cases there is a tendency to shortness of breath, rapid heart action, and bronchial cough, which is greatly exaggerated by physical exercise. Well-marked anemia may also be present. In many instances the above symptoms are so slight as to be almost or quite overlooked, but the physical signs are very characteristic. The apex beat, which is distinct and forceful, is found outside of the nipple line in the fifth intercostal space. Auscultation reveals a blowing systolic murmur most intense at or near the apex, but very distinctly transmitted to the left below the axilla in the direction of the lower portion of the scapula. It is one of the peculiarities of this condition in childhood that this murmur may be heard almost as distinctly in the back, between the lower spine of the scapula and the vertebral column, as it is in front. The second sound of the heart is usually accentuated over the pulmonary area, but if it be sharply and intensely reduplicated it is an unfavorable sign, indicating a severe mitral lesion with increased blood pressure in the pulmonary

artery. An increase in the size of the heart downward and to the left may be demonstrated both by percussion and by a radiograph.

**Mitral Stenosis.**—Mitral stenosis is a much less common but a much more serious lesion in childhood than mitral insufficiency. It is usually caused by repeated attacks of a low grade of rheumatic endocarditis. The history of rheumatism elicited in these cases is of the milder and more chronic types, and in some cases it is entirely wanting. The disease is, as a rule, rather insidious in its onset, and, during its early stages, especially in younger children, some of its characteristic physical signs may be absent. Jacobi has especially called attention to the fact that mitral

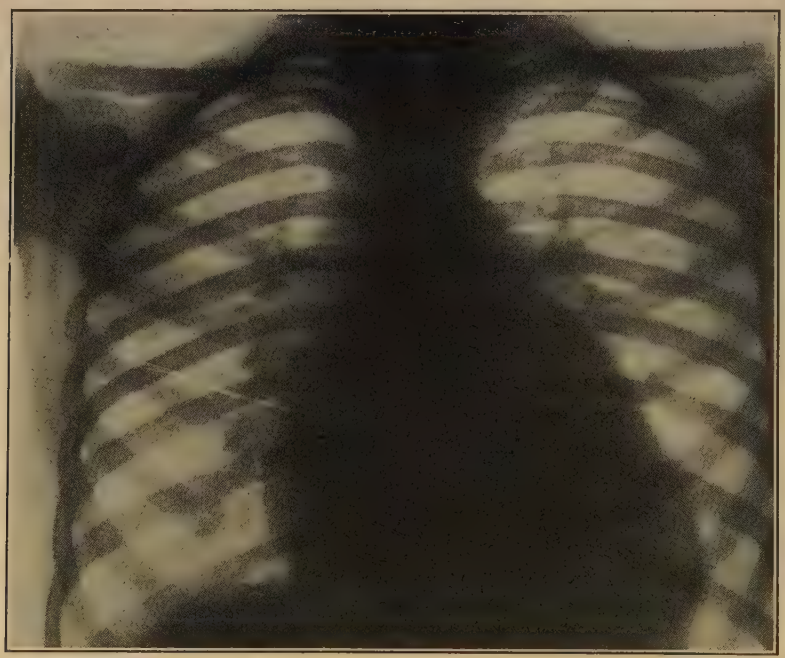


FIG. 80.—ENLARGED HEART FROM MITRAL REGURGITATION. (S. Lange.)

stenosis may exist without the presence of a diastolic murmur. This condition is rare in early childhood, but is much more frequently seen after the tenth year. It may exist as an independent condition, or it may be associated with mitral regurgitation. Poynton and others note the fact that a systolic bruit may precede and then give way to a diastolic or presystolic bruit as mitral stenosis becomes well established. In mitral stenosis the auricle is called upon to force its blood stream through a contracted mitral opening, and, as a result, it undergoes dilatation and hypertrophy. The blood is dammed back into the pulmonary circulation and the blood pressure in the pulmonary artery is greatly increased. Rapid and sometimes extensive hypertrophy of the right ventricle results. This hypertrophied right ventricle may continue for a long time to force suf-

ficient blood through the pulmonary circuit to supply the demands of the general circulation which is kept up by the left heart, but even under these favorable conditions the left ventricle does not receive enough blood to supply the general circulation, and, as a result, this ventricle, instead of undergoing hypertrophy, as in mitral regurgitation, may become smaller or remain the same size, so that the apex beat in this condition is not displaced. With failing compensation the right ventricle becomes dilated and the general symptoms of cardiac insufficiency develop.

Symptomatology.—This condition is commonly insidious in its onset. Lack of development, anemia, irritable heart, and, as Chapin has observed, pain in the region of the heart and dyspnea on exercising are usually present.

On auscultation a characteristic rough, presystolic murmur immediately precedes and is sharply terminated by a snapping first sound at the apex. Its point of greatest intensity is over the apex of the heart, which is found in its normal position. The murmur, unlike that of mitral regurgitation, is not transmitted toward the axillary line. The pulmonic second sound is accentuated. Increase in the size of the heart, as shown by percussion or by a radiograph, may be marked, and extends upward and to the right beyond the sternum. A presystolic thrill may be felt over the heart. Where mitral insufficiency is associated with mitral stenosis, as it is in a large percentage of these cases, there is a combination of the physical signs of the two conditions, with a double murmur at the apex, the diastolic immediately preceding the systolic murmur.

**Aortic Regurgitation.**—Aortic regurgitation is rare, but is seen in later childhood. Its characteristic sign is a diastolic murmur, having its point of maximum intensity in the second, third, or fourth intercostal space near the sternum. It is transmitted downward along the sternum. The heart is greatly enlarged, due to hypertrophy of the left ventricle. The apex beat is displaced downward and outward, and the cardiac dullness is increased in the same direction. The greatly hypertrophied left ventricle drives with great force the blood stream into the aorta and with the recoil of this stream, due to the defective valve, we have the "water-hammer pulse," characteristic of this condition. This is the most serious of cardiac lesions, usually resulting fatally before or about the time of puberty. The symptoms of cardiac insufficiency are much more commonly present than in mitral lesions. Pallor, stunted growth, rapid, irregular pulse, dyspnea, and cyanosis are common, and, with the breaking down of the partial compensation, the symptoms of gradual cardiac failure appear. Sudden death may occur in these cases.

**Aortic Stenosis.**—This is also a rare condition and is not uncommonly associated with aortic regurgitation. The characteristic bruit is a harsh, systolic murmur heard most distinctly in the second right interspace near the sternum and is transmitted upward into the large arteries of the neck. The hand on the chest feels a distinct systolic thrill. The left ventricle is hypertrophied, the apex beat is displaced downward and outward, and

an increased dullness may be outlined in the same directions. The symptoms of cardiac incapacity are the same as those above noted in aortic regurgitation.

**Tricuspid Regurgitation.**—Tricuspid regurgitation occurs in congenital conditions and as a late development of failing compensation in mitral and aortic disease. Apart from these conditions it is almost unknown in childhood. It is characterized by a systolic murmur heard over the lower portion of the sternum.

**Failing Compensation.**—Failing compensation may result from the seriousness of the valve lesion itself, from too severe physical strain thrown upon the heart, or from repeated attacks of endocarditis. In this condition the blood is dammed back into the pulmonary circulation, producing a great increase in the blood pressure of the pulmonary artery, which causes a sharp, quick closing of this valve, reduplication of the second sound, and later incompetency of the tricuspid valve. It will thus be seen that the giving way of the heart in chronic valvular disease may first be indicated by a marked accentuation and reduplication of the second sound over the pulmonary valve, and later by a marked dilatation of the heart with a displacement of the apex beat downward and outward. This great increase in the size of the heart, which occurs in failing compensation, results in the development of other murmurs, associated with the aortic, and possibly the pulmonary, valves. The great enlargement of the heart in this condition may be seen in a radiograph, or made out by percussion. Associated with the great increase in the area of cardiac dullness, the apex beat is diffuse and the vibrations of the struggling heart may be seen and felt in the intercostal spaces. In these cases the pulse beat is irregular and rapid; dyspnea, orthopnea and cyanosis are present. The legs may be slightly swollen, and in some instances a general anasarca may develop, the abdominal cavity being filled with fluid. This gradual failure of compensation, resulting in death, is unusual in childhood. It much more commonly occurs at or after puberty.

**Prognosis of Chronic Valvular Disease.**—The prognosis of chronic valvular disease depends upon the following conditions: first, the valve affected; second, the extent of the valvular lesion; third, the completeness of the resulting compensation; fourth, the possibility of preventing recurring attacks of endocarditis; fifth, the proper regulation of the life of the child, so that he may have proper exercise without throwing severe strain at any time upon the heart muscle.

The prognosis is best in uncomplicated mitral regurgitation. In many of these cases compensation may be so complete that the heart is physiologically competent to carry on the circulation under the ordinary conditions of life, so that these individuals may be useful members of a community throughout a long life. In mitral stenosis the prognosis is usually not so good, and there is more danger that the heart, even in the favorable cases, may show a failing compensation at or about puberty. In aortic disease the prognosis is, as a rule, bad, as few of these cases live to

be useful members of a community; they commonly die in later childhood or about puberty. Where more than one valve is affected the gravity of the prognosis is greatly increased.

The extent of the valvular lesion can commonly be estimated by the displacement of the apex beat, the degree of cardiac hypertrophy and dilatation, and by symptoms pointing to increased blood pressure in the pulmonary artery. The gravity of the prognosis is in direct proportion to the severity of these signs and symptoms.

When complete compensation has been established and all symptoms of cardiac irritation have subsided and the child is able to resume his ordinary vocations without cardiac distress, the prognosis is good, and it is correspondingly bad when, after proper and prolonged treatment, complete compensation cannot be established.

**Treatment of Chronic Valvular Disease.**—The most important factor in the prognosis, in those cases in which compensation has been established, is the possibility or probability of preventing recurring attacks of endocarditis. In accomplishing this end the cause of the previous endocarditis must be taken into consideration. In the great majority of these cases rheumatism is the exciting factor, and the treatment, therefore, largely consists in the prevention of subsequent attacks. These children should, if possible, for a number of years avoid the cold, damp, changeable weather which prevails during the winter months in our middle and northern States, by spending the months of January, February and March in a warm, dry, and equable climate. Those children that are compelled to remain under unfavorable climatic conditions should, during the winter months, wear woolen underclothing, sleep in well-ventilated apartments, and in suitable weather spend a great portion of the day out of doors, being always careful to avoid damp cold. It is especially important that they should be properly shod so that their feet may be always dry and warm. Above all, they should not be allowed to go to school or be closely housed with other children where the air is bad and contagions are present. All diseases that in any way involve the throat and respiratory passages are especially dangerous, and every effort should therefore be made to protect them from contagious diseases, especially influenza and tonsillitis.

In the treatment of chronic valvular heart disease it is especially important to remember the close relationship which exists in many of these cases between tonsillitis and recurring endocarditis. Every child with a compensated heart lesion should have the tonsils and other lymphoid tissues in his throat and pharynx carefully inspected. If the tonsils and adenoids be enlarged or diseased, they should be removed during the spring or summer months, and the adenoid ring, of which they are a part, should thereafter be kept in as healthful a condition as possible, to prevent the entrance of the contagion of rheumatism and other acute infections.

Avoidance of heart strain is a most important measure in preventing the breaking down of compensation. To accomplish this the child must

be under such proper medical supervision that he may have prescribed for him the exercise necessary to the development of the heart without producing cardiac strain. The careful medical observer can, as a rule, tell the child what he should do in the way of physical exercise, but he is not always so fortunate as to have his directions carefully followed. The heedlessness of childhood and ofttimes the carelessness of parents are factors over which he has no control. The importance of this subject demands that he should give explicit directions, and not vague statements, with reference to exercise, and that he should impress upon the parents, as well as the child, the absolute necessity of following directions. No general rule as to the kind of sports and the character of exercise can be made which will apply to all cases of heart disease; each case must be studied individually and prescribed for accordingly. If the lesion be a mitral one and complete compensation, without great enlargement of the heart, has followed, it is probable that a return to the ordinary sports of childhood may be gradually accomplished without injury to the heart. Golf, horseback-riding, swimming, a moderate amount of ball-playing may in time be safely indulged in, but at no time, even in these favorable cases, should tennis playing, hard bicycle riding or endurance contests of any kind, such as running, rowing or rope jumping, be permitted. It should be remembered, moreover, that it is only the most favorable cases of fully compensated mitral insufficiency that can be allowed this freedom in outdoor sports. In most of the cases the exercise should be much more restricted throughout childhood, and the parents as well as the patients should be told that the greatest danger of failing compensation occurs at or about puberty, and that for this reason the heart muscle must be kept in as good a condition as possible, by gentle exercise and by avoiding heart strain, that this critical period may be safely passed.

The general regulation of the life of the child is important. Nutrition must be especially looked to, by giving suitable food at regular intervals. An excess of sweets is to be avoided; sugar in the form of candy, confections, and pastry may do much harm. The diet should be a general one consisting of vegetables, eggs, milk, cereals, fruit, and a moderate amount of meat. Disturbances of digestion, and constipation, should be carefully guarded against. Severe nervous and mental strain, such as may be associated with school work, is to be avoided; these children are easily excited, and their nervous systems readily break down from overwork and excitement. They require more rest than the normal child; they should go to bed early, and during the day, if there be the least sensation of fatigue, they should be required to lie down after their midday meals; this applies even to those cases where compensation is apparently complete.

Systematic medical treatment may be important. This applies especially to those cases in which rheumatism is the etiological factor. It consists largely in the giving of alkalies and the salicylates during the winter months for a number of years following the initial attack of endocarditis. Bicarbonate, or benzoate, of soda, 5 to 10 grains, may be com-

bined with (wintergreen) salicylate of soda, 3 to 5 grains, put up in a palatable vehicle. This should be given for one week in every month, and during the remaining time the child should drink alkaline waters.

If anemia is present, iron, arsenic, and cod-liver oil may be of benefit, but the presence of this symptom usually indicates an incomplete compensation, or the occurrence of some complicating disease, and in either event its cause demands careful study, and the resulting treatment will depend upon the cause. If well-marked anemia occurs, the child should be put to bed the greater portion of the day and night in the fresh air out of doors, or with the bedroom windows wide open; this, with careful feeding, and the above-named tonics, will produce a rapid improvement in the blood state.

*Treatment of Incomplete or Failing Compensation.*—In the foregoing sketch of the treatment of chronic heart disease with full compensation, no mention has been made of the use of digitalis or other heart tonics, because they are not indicated when compensation is complete. Great harm may be done by the unnecessary administration of these drugs to a patient whose heart muscle is fully capable of doing its work; a heart murmur is, therefore, not always an indication for heart stimulants. When the symptoms of incomplete or failing compensation are present, these heart stimulants, properly administered, in association with rest in bed, are, as a rule, necessary, but above all I wish to impress the fact that in these cases rest in bed should be the first order, and heart stimulants the second. It is a fatal mistake to try to overcome the rapid, irregular pulse and shortness of breath of children with chronic heart disease, by giving them digitalis and allowing them to remain upon their feet. Rest in bed is the all-important remedy, just as it is in acute cardiac affections. During the time they are confined to bed, and for some time thereafter, heart stimulants are of value. The best of these is digitalis; it may be given in the form of the tincture, from 3 to 6 drops, depending upon the age of the child; care, however, being taken that its administration does not disturb the stomach. The fat-free tincture may, as a rule, be administered in essence of pepsin over a long period of time without producing gastric disturbance. In some cases it may be necessary to substitute teaspoonful doses of the fresh infusion of digitalis, or from 3- to 5-drop doses of the tincture of strophanthus. The latter remedy, while not as reliable as digitalis in bringing about compensation, is of much more value in the child than it is in the adult, and serves a very useful purpose in those cases where digitalis disturbs the stomach. It should also be remembered that the action of digitalis is cumulative, and for this reason its administration should be interrupted from time to time, to be again resumed when the heart begins to flag. Sulphate of strychnin, 1/100 to 1/200 of a grain, may be of value as a respiratory, cardiac and general tonic, if given over a considerable period of time. In those cases that do not respond fully to the rest and digitalis treatment, and which stop slightly short of full compensation, the home administration of the Nauheim bath, which is

of such value in the treatment of myocardial insufficiency in the adult, may be tried. These baths are especially applicable in older children, and the rules governing their administration should be the same as those outlined in the treatment of myocardial insufficiency in the adult. In those unusual cases of chronic myocardial insufficiency associated with well-marked dropsy the amount of fluid taken by the patient should be restricted, if possible, to one quart in the twenty-four hours. In these cases all the fluid taken should be carefully estimated, so that the sum of all the water and liquid foods should not exceed this amount.

If marked ascites is present, paracentesis gives great relief, and is followed by improvement in the action of the heart. In this operation the same precaution should be taken as in the adult, the fluid being slowly withdrawn and the abdominal wall subsequently firmly supported by an abdominal bandage.

## CHAPTER LVIII

### FUNCTIONAL CARDIAC DISORDERS

Disturbances in the rate and rhythm of the heart's action, not associated with inflammatory disease of this organ, are common. The child, by reason of the immaturity and instability (lack of inhibition) of its nervous system, is normally predisposed to functional disorders of the heart, and this predisposition may be greatly increased by a neurotic inheritance and by anemia and general malnutrition. In children of this type the cardiac nervous mechanism is easily disturbed by reflex and toxic factors, such as are commonly present in gastrointestinal disturbances and the acute infections.

**Arrhythmia.**—Arrhythmia, or irregularity in the heart's action, is common in infancy and childhood, and has, as a rule, little pathological significance. It may occur in nervous children even during sleep, from slight or unknown exciting causes. It may result from fright, anger or nervous excitement of any kind; a cold bath, severe exercise, slight fevers, and bacterial intoxications may produce more or less irregularity in the rhythm of the heart's action; a variation of 20 to 30 beats may occur within a few minutes without special pathological significance. Palpitation of the heart, associated with pain in the side and shortness of breath on exercise, is not uncommon in nervous, anemic, rapidly growing children. As elsewhere noted, it is not improbable that the explanation for the association of certain cardiac neuroses in older children with their rapid growth may be due to the excessive action of the thyroid gland during this period of life, the increased function of this gland being responsible in part, at least, for both the rapid growth and the cardiac irritability.

**Paroxysmal Tachycardia.**—Paroxysmal tachycardia may occur in older children. These attacks are commonly produced by auto- or intestinal

toxins. They are not infrequently associated with constipation, coated tongue, bad odor to the breath, headache, perspiration, and sometimes with an elevation of temperature. A gouty or migrainous diathesis may be etiologically related to these cases, and masturbation may be a predisposing factor. I have seen this symptom group associated with attacks of recurrent coryza.

**Bradycardia.**—Bradycardia, or slow heart action, may also occur as a purely reflex disorder in nervous, malnourished, neurotic children. The exciting cause in these cases is commonly of intestinal origin. The association, however, of bradycardia and arrhythmia occurring in certain of the acute infections, such as diphtheria and influenza, may be of ominous significance, denoting a toxic myocarditis rather than a simple functional disturbance.

**Functional Heart Murmurs.**—Accidental and functional heart murmurs are very common in early, as well as in late, childhood. They very often have no apparent pathological significance; they may be hemic, due to profound anemia; they may be myocardial, resulting from malnutrition, irritation, defective innervation, or inflammation of the heart muscles, or they may be cardiopulmonary in character.

The most common accidental murmur is the "late systolic pulmonary murmur," spoken of by many writers and carefully described by Hamill and le Boutillier. It is soft-blowing in character, moderately high pitched, and is continuous with, or immediately follows, the first sound. This systolic murmur has its point of maximum intensity in the second left interspace close to the sternal border, but it may be distinctly heard lower down in the third and fourth spaces between the midclavicular and parasternal lines. It is best heard in the recumbent position, at the end of inspiration; it may entirely disappear upon forced inspiration, and is exaggerated by exercise. The position of the apex beat of the heart is not altered, and the area of heart dullness is not increased. It may be transmitted to the vessels in the neck and is usually associated with a venous hum over both sides of the neck. Hamill says that this murmur has no definite pathological significance. It may occur in the absence of anemia, but "unquestionably the conditions giving rise to this murmur are frequently associated with anemia." Lüthje, Hamill, and le Boutillier found this murmur in over 60 per cent. of institutional children under five years of age; it is therefore of the greatest importance that it should be carefully differentiated from bruits produced by organic disease of the heart. In making this differentiation it should be remembered that it is *not congenital* and that it is aggravated by the recumbent posture, is loudest at the end of expiration, and commonly disappears when the lungs are fully inflated. The location of the murmur, the normal position of the apex beat, and the normal area of cardiac dullness are most important.

Forchheimer has especially emphasized the fact that mild forms of acute myocarditis occurring in scarlet fever, diphtheria, typhoid, rheumatic fever, variola, gonorrhea, septicopyemia, acute nephritis, and other

acute infections characterized by fever may produce transient, systolic, apical bruits which disappear when the myocardium recovers from the irritation produced by the acute toxemia. Systolic bruits from this cause have their point of greatest intensity at the apex, and are, as a rule, not transmitted to the axillary line. It is most important to keep in mind the conditions under which this character of heart bruit may occur, and it is always wise in these cases to withhold a definite prognosis until the condition of the heart may be studied after the acute intoxication has disappeared, since in many of these cases it will be found that what seemed a simple myocardial bruit is later found to be due to a true endocarditis. This type of cardiac murmur is not infrequently associated with chorea.

The cardiopulmonary murmur produced by the movement of the air in that portion of the lung which is in direct contact with the heart is systolic in time and heard with maximum intensity at the apex along the left border of the heart. It is inconstant, heard loudest at the end of expiration, and is comparatively infrequent, especially in the young child.

Venous murmurs occur both in infancy and childhood. They are most commonly heard over the large veins of the neck; they may be associated with anemia, glandular tuberculosis, enlarged thymus, rickets, and other malnutritions. The venous murmur described by Eustace Smith has long been recognized; it is produced by the pressure of enlarged glands upon the innominate veins, and is made much more distinct by throwing the head of the child backward; by this movement the enlarged glands, back of the veins, push them forward and compress them against the manubrium sterni.

In the diagnosis of functional murmurs, it is important to remember that diastolic bruits are nearly always organic, and that, as Forchheimer says, "The accentuation of the second pulmonary sound is of little value for diagnostic purposes, first, because it so frequently occurs in children who have no heart disease, and, secondly, it exists in both organic and functional valvular conditions in older children."

**Prognosis of Functional Cardiac Disorders.**—The prognosis in purely functional endocardial murmurs is good. The prognosis in venous murmurs will depend upon the exciting cause.

**Treatment.**—Severe exercise is to be prohibited; moderate exercise out of doors is of great value in restoring the tone of the heart muscle. The malnutrition, anemia and nervousness in these cases is to be combated by living and sleeping out of doors; by a carefully regulated diet of easily digested foods, including milk, eggs and meat; and by such tonics as iron, arsenic and cod-liver oil. The individual case in every instance must be studied to determine the important underlying causes of the cardiac neurosis. In many instances constipation and gastrointestinal toxemia are to be carefully combated. In others the anemia requires treatment. In others some profound constitutional disturbance, such as tuberculosis, may have to be combated. While the curative treatment is being directed toward the removal of the underlying causes, it may be necessary for a time

to give nerve sedatives, such as the bromides or valerian, and in rare instances digitalis may be demanded.

## CHAPTER LIX

### PERICARDITIS

Pericarditis is an inflammation of the pericardium, which in childhood is usually associated with endocarditis, the two conditions having very much the same etiological factors.

**Etiology.**—It may occur *in utero,* is not uncommon during the first year of life, and is met with in increasing frequency throughout childhood. Acute rheumatism is the most common direct etiological factor; rheumatic polyarthritis and chorea are associated with a large percentage of the cases. Scarlet fever, sepsis, pneumonia, tuberculosis, and other acute and chronic infections may be exciting causes. Pericarditis may be produced by the transference of the infectious material through the blood or lymph channels, or by direct infection from contiguous diseased structures in the lungs, pleura or heart itself. Hochsinger says it is a peculiarity of the pericarditis of childhood that in infancy it depends chiefly upon pyemic infection, in early childhood mainly upon the spread of inflammatory processes, and in later childhood upon rheumatism, which may be associated with chorea; the exudate, therefore, in infancy is usually purulent; in early childhood generally serofibrinous, and in later childhood almost always purely fibrinous. The microorganisms most commonly found are pneumococci, streptococci, staphylococci, tubercle, colon and pyocyaneus bacilli.

**Pathology.**—The pathological anatomy is similar to that found in the adult. There are three varieties. The fibrinous variety is characterized by a fibrinous exudate covering both the visceral and parietal pericardium, the rough surfaces of which are rubbed together by the action of the heart, producing the to-and-fro friction rub. The serofibrinous variety is the same as the above, with the addition of a serous exudate, which as it collects gradually separates the roughened surfaces of the pericardium; in some instances there may be an enormous dilatation of the sac. These two varieties are commonly but different phases of the same pathological process, and rheumatism is the all-important etiological factor. In the purulent variety the exudate is composed of pus or seropus, which may be tinged with blood. In some of these cases miliary tubercles may infiltrate the pericardium, in others the disease may be associated with pneumonia or purulent pleurisy. Again, a simple serofibrinous pericarditis may be converted into the purulent form. Endocarditis and myocarditis are very commonly associated with pericarditis. With the absorption of the fluid and the subsidence of the inflammation, adhesions may occur between the pericardial layers, which greatly cripple the action of the heart. Rarely in

mild forms of pericarditis, due to rheumatism, there may be complete restoration of the parts.

**Symptomatology.**—Pericarditis is frequently a very obscure condition, and its recognition is ofttimes difficult; in many instances the diagnosis is made on the post-mortem table. Mistakes in diagnosis may be due to the fact that there is a complicating endocarditis or myocarditis, and the symptom group is thereby confused, but in most instances they are due to lack of careful physical examination. The general symptoms, while not characteristic, are important and suggestive. Fever is nearly always present; in the fibrinous and serofibrinous varieties it may not rise above 101° or 102°F. In the purulent form it is remittent or intermittent in character, running as high as 104° or 105°F., and perhaps falling within the day to normal or below; this type of temperature, associated with other signs of cardiac disease, commonly means either a septic endo- or pericarditis, and the physical signs must make the differentiation. In pericarditis there may be and commonly is palpitation, precordial pain and more or less marked dyspnea, sometimes amounting to orthopnea; the pulse is rapid, frequently reaching 130 or 160, and not infrequently cyanosis and marked acceleration of the respiratory movements are present. The above symptom group should suggest pericarditis, rather than a simple endocarditis; the same symptom group, however, may be present in myocarditis with acute dilatation. It should be remembered, however, that not every case of pericarditis is marked by the above symptoms of cardiac distress; in some instances the disease is very insidious in its onset, and may not be suspected until physical signs reveal its presence.

Physical Signs.—A to-and-fro friction rub, synchronous with the heart's action, is the most typical sign of this disease. In the beginning it is usually soft and later becomes hard and grating; it is commonly heard best over the base, and firm pressure with the stethoscope may make it more distinct. Change of position may cause it to vary in intensity; it is least distinct with the child lying upon its back, and is commonly exaggerated when the child sits up or leans forward. In most instances it lacks constancy and does not continue to be synchronous with the to-and-fro action of the heart; it may be heard only during systole, or complete intermittency may occur. As effusion occurs it becomes less and less distinct, finally disappearing altogether as the layers of the pericardium are separated. In these cases the rub may sometimes be again discovered and the apex beat again be felt by placing the child in the knee-elbow position. With the disappearance of the friction sound, due to the increase of exudate in serous and purulent pericarditis, the heart sounds are muffled, the apex beat becomes fainter, more diffused and may entirely disappear. With the absorption of the exudate, the heart sounds again become more distinct, the friction rub returns to remain, for a time, until convalescence is fully established and recovery has resulted either in complete absorption of the fibrinous exudate or in adhesions between the pericardial layers.

Where the pericardial effusion is great, inspection may reveal a bulging

over the cardiac area, and percussion shows a very great increase of the cardiac dullness, which may extend as high as the second rib, an inch to the left of the sternum, curving from that point, well outside the nipple line and normal apex beat, as low as the sixth or seventh rib. To the right the dullness may extend 2 cm. or more beyond the sternum, and, continuing downward, become continuous with the liver dullness. The outlines of the distended pericardial sac are clearly shown by radiography.

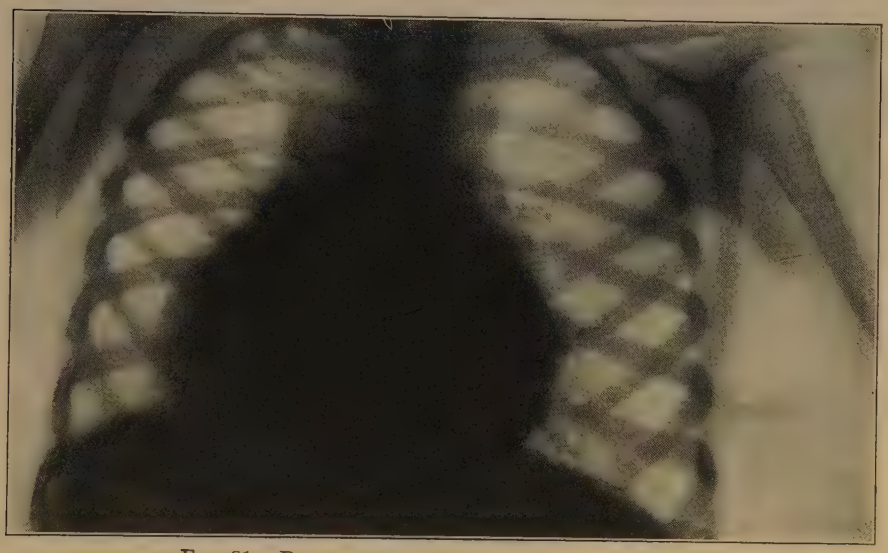


FIG. 81.—PERICARDITIS WITH EFFUSION. (S. Lange.)

In fatal cases the anemia deepens to a marked pallor, the dyspnea becomes an orthopnea, the cardiac pain is more marked, the patient is restless, sleepless, and vomiting occurs. The area of cardiac dullness may increase enormously, the liver becomes enlarged, the pleura fills with fluid, the urine is scant and albuminous, the pulse is rapid, irregular and flickering. Somnolence and coma may end the scene.

**Diagnosis.**—There should be little difficulty in differentiating endocarditis from pericarditis, except in those cases where the two conditions occur at the same time. In endocarditis the physical signs are constant and little influenced from day to day by changes in the condition of the heart, while in pericarditis the variability in the physical signs, from day to day, is noteworthy. The murmurs in these two conditions differ in their character, location and point of intensity, and differ especially in the fact that the murmur of endocarditis is transmitted, while the friction rub of pericarditis is not conducted, but is confined largely to the base of the heart and always to the pericardial region. The differential diagnosis, however, between pericardial effusion and acute dilatation of the heart is ofttimes a matter of great difficulty. The pericardial rub being absent, both conditions may present the marked increase in the area of cardiac

dullness, with the symptoms of cardiac distress. In acute dilatation, however, these symptoms appear more rapidly, and in this condition also, palpation over the cardiac area reveals a cardiac thrill, and the apex beat, while faint and diffused, can readily be located by placing the patient in the knee-elbow position.

**Prognosis and Course.**—The prognosis in pericarditis varies greatly with the individual case. The purulent cases with a septic temperature curve are, as a rule, fatal; a small percentage may recover if the pericardial sac is opened and carefully drained. In non-purulent pericarditis the prognosis, so far as life is concerned, is good, but in most of these cases the heart is permanently crippled by extensive adhesions. On the whole, therefore, it should be recognized that pericarditis of any form is a very serious disease, and that, while complete recovery may occur in a few of the mild cases, it is not to be expected. From the standpoint of etiology the pericarditis, associated with acute rheumatism and chorea, has a much more favorable prognosis than that produced by or associated with other infections.

The duration of the acute symptoms may vary from one week in the mild, to many weeks in the severe, cases; in the average, the physical signs run their course in about three weeks, but there is a long period of weeks, and sometimes months, during which the heart makes a slow, and in the majority of instances, only partial, recovery.

**Treatment.**—The PROPHYLACTIC TREATMENT of pericarditis is the same as that of endocarditis. The child should be protected from rheumatism, and second attacks of this disease should be prevented if possible. Diseased tonsils and adenoids should be removed in all children who have suffered from one attack of rheumatism. When rheumatic symptoms occur, the child should be placed in bed, the disease carefully treated, and the heart watched for evidence of acute inflammation.

TREATMENT OF THE ATTACK.—Absolute rest in bed is to be insisted upon throughout the acute symptoms; the patient during this time should do nothing for himself that can be done by others. Following this, "the rest-in-bed" treatment, somewhat modified to suit the exigencies of the case, should be continued until the heart has sufficiently recovered its physiological competency to permit the patient to get out of bed without producing symptoms of heart strain. His diet should be simple, nutritious and suited to his digestive capacity; milk, eggs and cereals are to be recommended. If rheumatism be present, it should be carefully treated as noted in the chapter on that disease. The antirheumatic treatment, however, should not be given after the acute rheumatic symptoms have disappeared, as it exercises no controlling influence over the inflammation of the pericardium. An ice-bag should be applied to the pericardial region and kept there the greater portion of the time, until the acute symptoms have commenced to subside. The ice-bag reduces the temperature, quiets the action of the heart and probably modifies the severity of the pericardial inflammation. Blisters and counterirritants over the region of the heart do more

harm than good. In chronic pericarditis 1 drachm of tincture of iodin, mixed with 1 ounce of anhydrous lanolin, may be used as an inunction over the cardiac region. Iodin given in this form is rapidly absorbed, and may possibly have some influence in promoting absorption. Morphin in from 1/10- to 1/50-grain doses may be given hypodermically to relieve pain; this is only necessary where the cardiac distress is very marked. If bromid of soda in 5- or 10-grain doses relieves the nervous irritability and cardiac distress, it may be used instead of morphin. Where the heart is weak and myocardial insufficiency threatens, strychnin, digitalis and whiskey should be given. The digitalis, especially in many of these cases, serves a useful purpose, but its action should be carefully watched, and if its administration is followed by an improvement in the action of the heart, it should be continued. Whiskey and strychnin, if they do not disturb the stomach, can do no harm and may, therefore, be given for days at a time, where cardiac and respiratory stimulation are necessary.

The treatment of pericarditis with marked effusion calls upon the physician to decide the difficult question as to whether an attempt shall be made to remove the fluid either by aspiration or by radical surgical measures. In the opinion of Rotch, "Paracentesis of the pericardium should unhesitatingly be performed when life is in danger from undue distention of the pericardial sac. A small aspirating trochar should be used. Opinions differ widely as to the best point of puncture. Inasmuch as the heart, when an effusion is present, remains in its usual position and does not, even when much enlarged, impinge on the fifth right interspace, and as the effusion, even when in so small an amount as 100 c. c., is found at that point, I consider it more rational to choose the fifth right interspace, 4 cm. (1½ inches) outside the right border of the sternum, as the point for tapping, thus avoiding all danger of injuring the heart. At this point the right internal mammary artery will not be injured. Another place to aspirate, recommended by Osler, is the left fourth interspace, either close to the sternal margin or 2.5 cm. (1 inch) from it, in order to avoid wounding the internal mammary artery. The left fifth interspace, 3.75 cm. (1½ inches) from the sternal border, may also be taken for the point of puncture." After introducing the needle as above directed, such fluid as will readily flow through the trochar should be allowed to drain away very slowly. And if this operation reveals a purulent pericarditis, the case should be referred to the surgeon for operative treatment, since these cases are practically hopeless under any other mode of treatment. In properly selected cases, especially in older children, exsection of the rib, and the opening and free drainage of the pericardial sac, gives to the patient almost the only chance he has for recovery.

## CHRONIC PERICARDITIS WITH ADHESIONS

This condition is commonly caused by rheumatism or tuberculosis. In rare instances there is no history of an acute attack, the disease being in-

sidious in its onset. Nearly all cases follow acute pericarditis; in the great majority pericardial adhesions are left, which permanently cripple the heart. The pericardium is thickened and its visceral and parietal layers are adherent, completely or partially obliterating the pericardial sac. Adhesions may also bind it to the diaphragm, pleura, or chest wall. A low grade of chronic myocarditis usually follows, resulting in increasing dilatation and increasing weakness of the cardiac muscle, and finally resulting in death from cardiac insufficiency. In other instances, especially in those cases of rheumatic origin, the cardiac muscle, forced to do extraordinary work, because of pericardial adhesions, becomes greatly hypertrophied. These cases may live for many years under proper medical supervision.

**Symptomatology.**—The symptoms, as a rule, are those of gradually increasing myocardial insufficiency; the pulse is rapid, weak, and irregular, easily influenced by slight exertion. Cardiac pain and dyspnea are usually developed by exercise; sudden death from cardiac insufficiency may occur at any time. In other instances there is slow failure of the cardiac muscle, lasting over months or years.

The most characteristic physical sign is a retraction of the chest wall, especially noticeable over the lower cardiac area, occurring with every systole. Immobility of the heart is a valuable sign; the position of the apex does not move with a change in the position of the child. There is an exaggerated diastolic shock accompanying the second sound over the greater part of the pericardium (Broadbent). Friction sounds may be heard, but, as a rule, are not present. The area of cardiac dullness is greatly increased and X-ray pictures show great increase in the size of the heart, and may show adhesions. The valve sounds, which may be present in these cases, may be due to chronic endocarditis or to dilatation, with a resulting valvular insufficiency; when present they confuse rather than assist in the diagnosis of adherent pericardium. Pericarditic pseudocirrhosis of the liver may occur (Pick's disease).

**Diagnosis.**—The diagnosis is very difficult if the characteristic sign of systolic chest retraction is not present; the diagnosis, however, may oft-times be inferred if the physician was fortunate enough to have seen and made the diagnosis of the acute pericarditis, which was afterward followed by the symptoms of myocardial insufficiency above noted.

**Treatment.**—The treatment of this condition is the same as that of the myocardial insufficiency, which occurs in chronic valvular lesions.

# SECTION IX

## *DISEASES OF THE BLOOD AND DUCTLESS GLANDS*

## CHAPTER LX

### THE BLOOD

**Red Blood Corpuscles** (*Erythrocytes*).—Normal adult blood contains 5,000,000 red blood corpuscles, 7,500 white blood corpuscles, and 200,000 blood plates to the cubic millimeter. Erythrocytes (red blood corpuscles) are the hemoglobin carriers, and the potency of their most important function, that of bearing oxygen from the lungs to the tissues, depends upon the amount of hemoglobin they contain. Hemoglobin is therefore the most important constituent of the blood. The specific gravity of the blood varies directly with the amount of hemoglobin it contains. The NORMAL ERYTHROCYTE (NORMOCYTE) has no nucleus and varies in size from 6 to 9 $\mu$. Larger and smaller forms are frequently met with. The small red blood corpuscles are known as *microcytes;* they may be less than half the size of the normocyte, varying from 5 to 3 $\mu$. The large red blood corpuscles are called *macrocytes* (9 to 12 $\mu$), *megalocytes* (12 to 16 $\mu$), and *gigantocytes* (16 to 20 $\mu$). All the above forms of red blood corpuscles are non-nucleated and may appear normally in the blood of very young infants. The very large and very small forms are pathological, except during the early days of life. They may be present in considerable numbers in all forms of anemia, but they have not the pathological significance of the NUCLEATED FORMS (ERYTHROBLASTS). The following varieties of erythroblasts occur: *Normoblasts* are nucleated red blood corpuscles of normal size. They are the immediate antecedents of normal red corpuscles, and occur normally in the blood of the embryo in large numbers; a few may be found during the first few days after birth, but soon disappear. Each normoblast contains a round, sometimes irregular, darkly staining nucleus, one-half the diameter of the cell. They are found in all forms of severe anemia, both primary and secondary. *Megaloblasts* (*gigantoblasts*) are red blood cells two or three times the normal size, containing a large, round, or irregularly shaped nucleus. The cytoplasm is frequently polychromatophilic, and those cells of irregular shape are called *poikiloblasts;* the presence of these corpuscles, especially in large numbers, indi-

cates a severe type of anemia. *Microblasts* are red blood corpuscles often less than half the normal size, containing a small, deeply staining nucleus. They are seen in primary and in severe types of secondary anemia.

*Poikilocytosis* is the term used for the distorted and irregularly shaped and sized red blood corpuscles; they occur in various types of anemia, especially the grave primary forms. In the more severe types these ill-shaped irregular forms are present in great numbers. By *anisocytosis* is meant a variability in size of the red corpuscles in a given specimen. *Oligocythemia,* or a scarcity of red corpuscles, is most marked in pernicious anemia and in the anemias of infancy and early childhood. *Polycythemia,* an increased number of red corpuscles per cubic millimeter, occurs frequently in the anemias of infancy and early childhood, especially of the chlorotic type, and also as a clinical entity in later life. In *polychromasia,* or *polychromatophilia,* the red cells show a varying affinity for basic dyes in addition to their normal reaction to acid dyes; this may occur in any form of the primary and secondary anemias, but is not marked in chlorosis. *Oligochromemia,* or a scarcity of hemoglobin per unit volume of the blood, is still more characteristic of the anemias of childhood than of adult life. The color index of the blood refers to the total amount of hemoglobin as related to the number of red cells. It is determined by dividing the per cent. of hemoglobin by the per cent. of red blood cells. The normal per cent. of hemoglobin in infancy is 58 to 78, and the normal per cent. of red blood cells is 100; the color index of the normal infant is therefore 70 divided by 100, or 0.70. In anemic conditions in infancy the normal low color index is still further reduced; that is to say, the anemia tends toward the chlorotic type, in which there is a greater reduction of hemoglobin than of red blood corpuscles.

**White Blood Corpuscles** (*Leukocytes*).—White blood corpuscles are represented by the following varieties: *Small lymphocytes* (*small mononuclear leukocytes*) are about the size of a red blood corpuscle, consisting of a large nucleus, surrounded by a narrow rim of cytoplasm. They are the predominating form in infancy and early childhood. During the first year of life they represent from 53 to 55 per cent. of all leukocytes, while in the adult they represent only about 24 per cent. There is a gradual diminution in the percentage of small lymphocytes throughout childhood. They are formed in the spleen, lymph nodes, and other lymphatic tissues. The small lymphocytes are notably increased in whooping-cough, measles, in severe forms of anemia, in the status lymphaticus, and in all conditions in which there is a hyperplasia of the spleen and other lymphatic tissues. They are enormously increased in the lymphatic form of leukemia. *Large lymphocytes* are two or more times the size of the small lymphocytes, and contain a large oval nucleus surrounded by a narrow rim of cytoplasm. The *large mononuclear leukocytes* (*splenocytes*) are two or three times the size of red corpuscles and contain a large single nucleus in a large amount of nongranular or faintly granular cytoplasm. The nucleus is frequently irregular, and when it shows a marked indentation the cell is called a

"transitional cell." These cells represent from 3 to 6 per cent. of the total number of leukocytes, although they are frequently much more numerous in infancy and early childhood. They are formed chiefly in the spleen. *Polymorphonuclear neutrophiles* are about 12 $\mu$ in diameter, a little less than twice the size of the red blood corpuscle. They each contain a chromatin-rich nucleus which is polymorphous, and may resemble the letters E, V, S, U and Z, or show wreathed or rosette forms. This nucleus is surrounded by a neutrophilic granular cytoplasm. In infancy they represent 35 per cent. of the total number of leukocytes, and gradually increase in percentage throughout childhood to from 60 to 70 per cent. in the adult. They are produced, as are other granular cells, by the bone marrow, and descend directly from the neutrophilic myelocytes. They are increased in diphtheria, pneumonia, scarlet fever, smallpox, meningitis (the tuberculous form excepted), rheumatism, and especially in all septic cases such as septicopyemia, septicemia, appendicitis, peritonitis, septic arthritis and acute inflammatory processes of all kinds; they are also found in great excess in myelogenous leukemia. *Eosinophiles* are generally slightly larger than the neutrophiles above described; the nuclei are usually bi-lobed, frequently tri-lobed and polymorphous; they differ from the neutrophiles also in having large refractive granules, which stain with acid dyes, such as eosin. They are formed by the eosinophile myelocytes in the bone marrow. In infancy they represent only 1 or 2 per cent., and in adults 1 to 4 per cent., of the total number of leukocytes. They are increased by diseases due to animal parasites, such as trichiniasis and uncinariasis, in chronic skin diseases, leukemia and other diseases of the bone marrow, postfebrile conditions, and in bronchial asthma. *Basophiles,* or *mast cells,* are granular cells slightly smaller than the neutrophiles, having a somewhat irregular, frequently knotted or tri-lobed nucleus; the granules, generally large, stain only with basic dyes. They are very scanty in normal blood, and a proportion of 1 per cent. or over is pathological. They are of diagnostic value in myelogenous leukemia, where they are greatly increased.

*Blood platelets* are small, probably non-nucleated forms, believed by some to be related to the white blood corpuscles, by others to the red corpuscles. They are of irregular shape, from 2 to 3 $\mu$ in diameter, and their average number is from 200,000 to 300,000 to the cubic millimeter, according to various observers. In pathological conditions their number may be less than 100,000 or more than 300,000. Further than that they are concerned in the formation of fibrin and in the clotting of blood, their physiological significance is not known. They are increased in posthemorrhagic and secondary anemias, pneumonia and tuberculosis, and especially in myelogenous leukemia. They are decreased in lymphatic leukemia, pernicious anemia, hemophilia, and in some forms of purpura.

Pathological White Corpuscles.—Myelocytes (marrow cells) are usually larger than the polymorphonuclears, but they may vary in size. They contain one large oval nucleus, surrounded by a rim of protoplasm containing granules, either neutrophilic or acidophilic. This cell occurs

very rarely in the blood of the new-born, and otherwise is found in the peripheral blood only in pathological conditions. It represents the immature (polymorphonuclear) leukocyte which has been hurried out from the bone marrow before its completion. Three varieties may occur: *neutrophilic myelocytes,* the granules of which stain with neutral dyes; *eosinophilic myelocytes,* the granules staining a brilliant red with eosin; *basophilic myelocytes,* the granules of which stain only with basic dyes. Myelocytes may be found in infancy in all conditions in which there is a marked leukocytosis. They are always present in large numbers and are of special diagnostic importance in myelogenous leukemia, and also in disease of the bone marrow and other blood-forming structures.

**Peculiarities of the Blood in Infancy and Childhood.**—The blood of infants differs materially from that of adults. It is an immature tissue, which gradually approaches the adult type. The morphological changes which take place in the blood throughout infancy and childhood are clearly portrayed in the accompanying tables. In fetal life both red and white blood corpuscles are produced by the same blood-forming organs, the liver, bone marrow, spleen, thymus and lymphatic tissues in general; but toward the end of embryonic life the liver, under normal conditions, loses this function, but still continues for a time to hold a reserve supply of iron, from which hemoglobin is made during the early days of postnatal life. After birth the marrow, spleen and lymphatic tissues continue to be the important blood-forming organs, and nature then makes the attempt to differentiate the special work of these organs, so that the red blood corpuscles and granular leukocytes (polynuclear neutrophiles, mast cells, eosinophiles and myelocytes) may have their origin exclusively in the bone marrow, and the lymphocytes exclusively in the spleen, lymph glands and other lymphatic tissues. This differentiation is almost perfectly established in the adult, but in the infant, under certain abnormal conditions, when there is a great demand made upon these blood-forming organs for either the production of red or white cells, there may be a return to the fetal conditions, in which both red and white cells are produced in all of the blood-forming organs. In the more severe forms of anemia the liver itself becomes enlarged and again assists in the blood-forming process. The greatest portion of this extra work falls, however, upon the spleen and lymphatic glands. These organs, especially the spleen, become greatly enlarged by a true hyperplasia, which occurs in response to the demand for this increase of function. This reversion of function is an explanation of the fact that in the anemias of infancy and young childhood there is a tendency on the part of the blood to revert to the infantile type; that is to say, the red blood corpuscles vary greatly in size, shape, and staining reaction, and nucleated forms are present. The blood in infancy, being in a formative stage, is much more vulnerable, and very great changes may be produced in it from comparatively slight causes. The blood-forming organs at this period are taxed to their full capacity under normal conditions, so that when any unusual call is made upon them, as in the severe

anemias of childhood, they are unable to meet the increased demands. This susceptibility of the blood to injury, and this lack of reserve power in the blood-forming organs, are the reasons why infants bear hemorrhage badly, and why such pronounced anemias develop from comparatively slight causes.

The marked deficiency of the blood-forming organs of the infant, and their lack of reserve power in the production of red blood corpuscles, do not hold true for white blood corpuscles. In infancy there is a normal leukocytosis, the variations and the special characteristics of which, throughout infancy and childhood, are detailed in the following tables. This leukocytosis is very readily increased by slight causes in the infant, so that little pathological significance attaches to a count under 20,000 in a child less than four years of age. After the sixth year the leukocyte picture approaches closely to the adult type, and counts made after this time of life have almost the same interpretation that they have in the adult. In the comparative study of the leukocyte blood pictures of the child and adult, it is important to remember that up to the sixth year of life the mononuclear cells or lymphocytes are relatively and actually greatly increased in numbers, and, since these cells are produced by the spleen, lymph glands, and other lymphatic tissues, their presence in increased numbers indicates an excessive activity of lymphatic tissues during this period of life. This accounts for the prevalence of lymphatic diseases in infancy and childhood, and also explains why the leukocytosis which occurs in the well-marked anemias of childhood as a result of disease or overwork of the blood-forming organs is commonly of the lymphocytic type. Digestive leukocytosis, however, which is very marked in infancy, and leukocytosis produced by general sepsis, are in the child, as in the adult, of the polymorphonuclear type.

The hemoglobin changes which occur in the blood of the infant and which are clearly portrayed in the following tables are of great physiological and pathological importance. The high percentage of hemoglobin which occurs at birth and which continues for a number of days is the continuation of the fetal condition, kept up by drawing upon the store of iron which the liver contains at this time. There is a sharp fall in hemoglobin about the third week of life, which is due partly to the exhaustion of the fetal supply of iron and partly to the dilution of the blood, which results from the large quantity of fluid taken by the infant. From this time on throughout childhood there is a slow increase in the amount of hemoglobin, but it does not approach the normal until the tenth year of life. During all of this time the child has a normal red cell count, so that each red corpuscle must be deficient in hemoglobin. In other words, there is a normal chlorotic condition, or low color index, which continues throughout early childhood. In all diseases which affect the blood or blood-forming organs, producing anemia, the hemoglobin suffers first and the chlorotic condition is increased. This is one of the marked characteristics of the anemias of childhood.

| Red Corpuscles | Premature | At Birth | 1st 24 Hours | 3d or 4th Day | 10th Day | 3d Week | 6th Month | 1st Year | 6th to 7th Year | 10th Year | Adult |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Number | | 6,000,000 | 7,000,000 to 8,000,000 | 6,500,000 | 5,000,000 | | 5,750,000 | 5,550,000 | 5,120,000 | 4,800,000 to 5,000,000 | 5,100,000 to 5,600,000 |
| Shape | | | variable | | variable | | slight variations | slight variations | occasional poikilocyte | occasional poikilocyte | |
| Size | | | anisocytosis | | anisocytosis | | slight anisocytosis | slight anisocytosis | | | |
| Staining reaction | | | | | | | | | | | |
| Hemoglobin | very high | 104 to 110 | 135 to 140 | 115 | 100 | 55 to 60 | 70 normally low | 74 | 80 to 90 | 90 to 95 | 95 to 100 |
| Color Index | | .85 to 9 | .85 to .95 | .88 | 1.00 | | .63 | .67 | .80 to .90 | .90 to .99 | .85 to .98 |
| Erythroblasts | preponderate | small number | small number | very few | none | none | none | none | none | none | none |

| White Corpuscles | Premature | At Birth | 7th Day | 6th Month | End of 3d Year | 6th to 8th Year | 10th Year | Adult |
|---|---|---|---|---|---|---|---|---|
| Number of Leukocytes | | 20,000 to 26,000 | 9,000 to 12,000 | 15,000 | 12,500 | Same as Adult | | 5,000 to 10,000 |
| Small Lymphocytes | | 15–18% | 18–30 | 50–70 | | 30–40 | 25–35 | 20–28 |
| Large Lymphocytes | | | | | | | | |
| Large Mononuclears and Transitionals | Mononuclears Predominate | 10–16% | | 6–14 | | 4–10 | 3–8 | 2–5 |
| Polymorphonuclears | | 70–75 | | 28–40 | | 45–55 | 55–60 | 60–72 |
| Eosinophiles | | | | 0.5–10.0 | | 0.5–5 | 0.2–4 | 0.5–4 |
| Mast Cells | | | | occasional | | 0.2–1.0 | 0.2–1.0 | 0.5–1.0 |

It will thus be seen that the blood picture in the anemias of infancy and early childhood cannot be interpreted by adult standards. These pictures may closely resemble pernicious anemia and leukemia in the adult and yet be due to causes that yield more or less promptly to treatment. The above tables will assist materially in the interpretation of blood pictures at different ages. The following blood changes are more or less characteristic of the anemias of infancy and young childhood. First, a marked deficiency of hemoglobin without a corresponding decrease in the number of red blood corpuscles, indeed frequently with an increased number of red corpuscles; this produces a low color index, or so-called chlorotic condition of the blood. Second, great variations in the size, shape and staining reaction of red blood corpuscles, and the presence of many

nucleated forms. Third, a leukocytosis, mild or severe, the nongranular cells or lymphocytes often predominating.

Enlargement of the spleen and other lymphatic tissues is very commonly associated with the secondary anemias of infancy. This association of splenomegaly and secondary anemia has been very generally utilized in the classification of these anemias.

## CHAPTER LXI

### SIMPLE SECONDARY ANEMIA

The simple secondary anemias are so called because they are secondary to some clearly defined disease or condition which causes destruction of or interferes with the formation of hemoglobin and red blood corpuscles. They are thereby distinguished from the primary and pernicious anemias, the causes of which are unknown, whose well-defined blood picture is, in the present state of our knowledge, considered to be due to some pathological factor which acts on the blood-forming organs. The blood pictures, however, presented by the secondary anemias of infancy and childhood are not so clearly differentiated from the primary anemias as they are in adult life. The embryonic type of blood contains a number of varieties of corpuscles which are considered more or less characteristic of the primary anemias of adult life, but which do not occur in the secondary anemias of the adult; while, on the other hand, in the secondary anemias of infancy and young childhood there is a tendency on the part of the corpuscles to revert to the embryonic type. For this reason they may present a blood picture containing the various varieties of nucleated red blood corpuscles and also red corpuscles varying greatly in their size, shape, and staining qualities, thus presenting a picture which may possibly be confused with the primary anemias. In addition to this, it is believed that the primary or pernicious anemias of early life may sometimes begin as simple anemias.

**Etiology.**—Simple anemia may be hereditary, the infant being born anemic and inheriting from diseased parents weak or defective blood-forming organs, which are unable to meet the demands of the rapidly growing body. Chronic intestinal toxemia and diseases of the gastro-intestinal canal are the most common causes of this condition in infancy. Chronic glandular tuberculosis is one of the most common causes of anemia in childhood, and where other causes are not apparent this condition is to be suspected. Among the other common anemia producers are non-tuberculous adenitis, rheumatism, malaria, syphilis, contagious diseases such as influenza and diphtheria, malignant disease, status lymphaticus, prolonged suppuration, chronic nephritis, intestinal parasites, loss of blood from hemorrhage, bad air, lack of sunshine and poor food. These latter

causes are potent factors in producing anemia among the poor of our large cities.

**Symptomatology.**—General Symptoms.—That the child is anemic is first made evident by the facts that the skin is pale, transparent or perhaps sallow, and that the mucous membranes gradually lose their color. The blanching of the skin and mucous membranes will vary with the potency of the underlying cause. In severe cases, especially in infancy, the skin may become edematous. The child lacks energy, is listless, has no appetite, and, as a rule, suffers from constipation and digestive disturbances. There is a gradual failure in health, the child losing in weight and strength. It has little endurance, is easily fatigued and suffers from shortness of breath on exercising. Nervous symptoms are always present; the child becomes irritable, sleepless, hysterical, frequently suffers from night terrors, headache, habit spasms, incontinence of urine, chorea, and other neuroses. In a large percentage of these cases the physician's attention is directed to the anemia in trying to discover the cause of a recently developed nervous syndrome.

In pronounced cases of anemia the heart is weak, rapid, irregular, and may be dilated. Hemic murmurs may frequently be heard over the base of the heart, and not uncommonly these murmurs, which are very distinct with the child lying upon its back, disappear when the upright position is assumed. The peripheral circulation is poor, the child is easily chilled, suffers from cold hands and feet. Slight enlargement of the spleen may be present without special pathological significance. The liver is also frequently increased in size.

Blood Examination.—From the above symptom group the diagnosis of anemia may be easily made, but the character and extent of the anemia must be determined by a blood examination. In interpreting the blood picture presented, the normal low hemoglobin percentage of infancy and early childhood must be kept in mind. There is an increased reduction of hemoglobin, and frequently a marked decrease in the number of red blood corpuscles, though in some cases the count may be nearly normal. The color index is low; that is to say, there is a proportionately greater decrease in hemoglobin than in red blood corpuscles, so that the anemia is mildly chlorotic in type. In severe cases the hemoglobin may be reduced to 25 or 30 per cent., and the red blood corpuscles may show a count of 4,000,000 to 5,000,000 or over, but in the very severe cases there is a great decrease, sometimes to 1,500,000. Irregularities in the shape and size of red blood corpuscles also occur, and nucleated forms are present. These are chiefly normoblasts, but in severe forms microblasts and megaloblasts may also occur. Leukocytosis frequently is present; this is commonly due to an increase in the lymphocytes, especially in gastrointestinal conditions; an increase in polymorphonuclears generally denotes some complication. If intestinal parasites be the cause of anemia, eosinophilia is marked.

**Diagnosis.**—In the vast majority of cases the diagnosis of secondary anemia can be made in the child, as in the adult, by the blood picture

alone. It is only in the very severe types where megaloblasts occur and polychromasia exists that the diagnosis may be in doubt, but even in these conditions the presence of an exciting cause, the great preponderance of normoblasts as compared with the megaloblasts, and the low color index should be sufficient to exclude pernicious anemia, a very rare disease in childhood.

In those cases associated with enlarged spleen and marked leukocytosis there may be great difficulty in deciding when the condition ceases to be a simple, secondary anemia, and becomes the pseudoleukemia of infancy (von Jaksch's disease). In this differentiation the blood picture of the latter disease will be of assistance.

**Prognosis.**—The prognosis, in the vast majority of cases, is good, since the secondary anemias of infancy and childhood, as a rule, depend upon removable causes.

**Treatment.**—Since secondary anemia is a symptom of some other disease, or is produced by remedial causes, the object of the physician should be to search carefully for the underlying causes and remove them. Fresh air, sunshine, good food, proper hygienic surroundings are important whatever may be the causative factor. Arsenic and cod-liver oil are valuable adjuncts in the treatment of almost all forms of anemia, and some easily assimilated form of iron is especially indicated, as it furnishes the material from which the blood-forming organs manufacture hemoglobin. Constipation and all abnormal conditions of the gastrointestinal canal must be carefully treated, and the child fed at regular intervals on a wholesome food suited to its digestive capacity.

## CHAPTER LXII

### PSEUDOLEUKEMIA OF INFANTS

### (*Von Jaksch's Disease*)

Under this term von Jaksch in 1889 described a rather clearly defined symptom group, characterized by the blood picture of grave secondary anemia, with leukocytosis, enlargement of the liver, spleen, and sometimes other lymphoid tissues. He believed this condition to be a distinct clinical entity, but the trend of opinion at the present time is that the cases grouped under this term are severe secondary anemias associated with enlarged spleen, occurring almost exclusively in infancy, and that age is largely the determining factor in the production of this type of anemia. The enlarged spleen, which is one of its distinguishing characteristics, is but one of the manifestations of the underlying pathological process, which is probably toxic in character. It is not to be considered as a causative factor.

**Etiology.**—Age is the all-important predisposing factor. The great

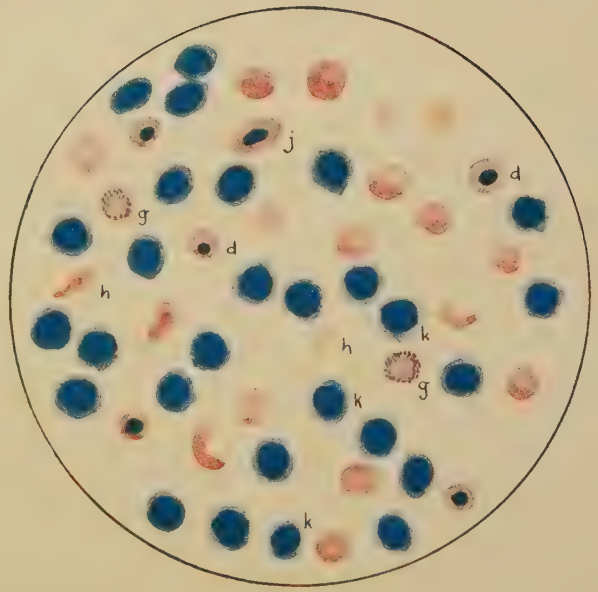



FIG. 1.—ACUTE LYMPHATIC LEUKEMIA.

a. Neutrophilic myelocytes.
b. Nucleated red cells with mitosis.
c. Polymorphonuclear neutrophiles.
d. Erythroblasts.
e. Mast cells.
f. Basophilic erythrocytes.

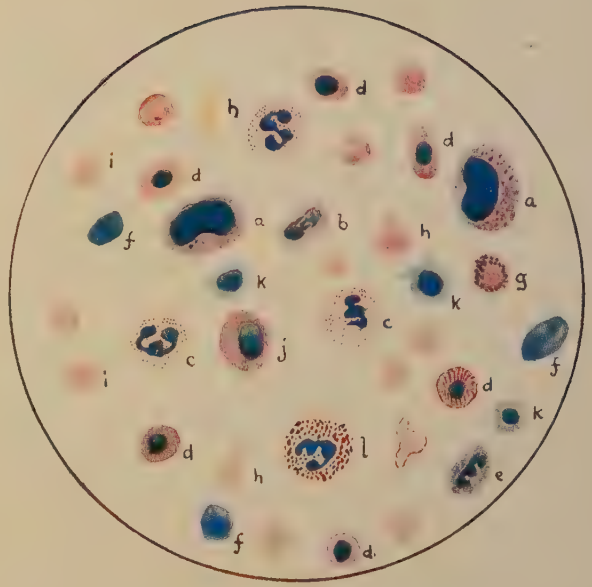



FIG. 2.—VON JAKSCH'S DISEASE.

g. Granular degeneration in red cells.
h. Poikilocytes.
i. Normocytes-erythrocytes.
j. Megaloblasts.
k. Lymphocytes.
l. Eosinophiles.

majority of these cases occur during the last half of the first year of life, but this disease may occur as late as the third or fourth year. The immaturity of the blood of the infant makes it especially vulnerable to toxic and other injurious influences, so that the normal blood picture at this age is easily disturbed by causes which later in life would have little effect upon the mature blood.

Chronic gastrointestinal intoxication associated with artificial feeding is the most important exciting factor. Unhygienic surroundings, diarrhea, constipation, chronic indigestion, syphilis, rickets, and secondary anemias are the usual antecedents of this condition. It is rare in breast-fed babies.

**Symptomatology.**—Blood Picture.—The hemoglobin and red blood corpuscles are markedly diminished, the former to 20 or 30 per cent., the latter to 2,000,000 or 3,000,000 to the c. mm.; in rare cases to under 1,000,000. The red corpuscles vary greatly in shape (poikilocytosis); small red cells (microcytes) and large red cells (megalocytes) are present, nucleated forms (normoblasts and megaloblasts) may be found, and polychromatophilia may occur. The white blood cells are increased, a leukocytosis of from 30,000 to 50,000 being common. Mononuclears and polymorphonuclears predominate, eosinophiles are commonly present, and neutrophilic and eosinophilic myelocytes may be found.

Other Symptoms.—Pallor of the skin and mucous membranes is very marked. The spleen is enlarged, hard, not tender to the touch. It may extend as low as the crest of the ilium, or may be felt but slightly below the edge of the ribs. Its size is not always in proportion to the severity of the other symptoms of this disease, yet it is the most distinguishing feature of the clinical picture. The liver is commonly slightly enlarged, and the superficial lymph nodes easily palpable. Slight hemorrhages into the skin and mucous membranes are common. An irregular fever may occur.

Obscure digestive disturbances should be carefully searched for. Constipated stools, mucous discharges, intestinal fermentation and indigestion are common, and indican is usually found in excess in the urine. The appetite is lost, the patient is listless and grows progressively weaker and more emaciated.

**Differential Diagnosis.**—From leukemia, the disease with which this condition is most commonly confused, it can only be differentiated by repeated blood examinations. The blood count in this condition shows few myelocytes and a moderate leukocytosis, while in leukemia the myelocytes are present in large numbers and the leukocytes enormously increased. In von Jaksch's disease the number of leukocytes seldom exceeds 50,000, and there is practically a sustained percentage of the various varieties of leukocytes in spite of the presence of the myelocytes. A large number of nucleated red corpuscles are commonly seen. In leukemia the liver and lymph nodes are much more enlarged.

From secondary anemias due to rickets, syphilis and other causes the diagnosis is made largely by the severity of the whole clinical syndrome.

The spleen is much larger, the leukocytosis more marked, and megaloblasts and myelocytes are more commonly seen.

Pernicious anemia rarely, if ever, occurs in infancy, and is not to be considered, therefore, in the differential diagnosis, although the blood picture of this condition may very closely resemble that of pernicious anemia in the adult.

**Prognosis.**—The majority of these cases recover under careful treatment. The condition, however, is always to be considered a grave one, and the prognosis should be carefully guarded until definite signs of improvement begin.

**Treatment.**—The indications for treatment are as follows:

First.—Remove the intestinal intoxication, which is commonly present. This comprehends the careful treatment of any abnormal gastrointestinal condition. If constipation be present, mild laxatives, such as milk of magnesia, may be used from day to day, with a dose of castor oil at intervals of four or five days. For a child one year of age, one grain of either salol, betanaphthol, or carbonate of guaiacol should be given every three hours as an intestinal antiseptic throughout the treatment. Great stress should be laid on the antiseptic treatment of these cases, even though gastrointestinal disturbances be not clearly evident.

Second.—Improve the infant's nutrition by selecting a diet within the range of its digestive capacity, and at the same time of such a character that it will serve nutritional purposes. Breast-milk is the best of all foods for this purpose, and many of these cases will rapidly improve if a suitable wet-nurse is secured. If wet-nursing be impracticable, these infants should be fed along the lines outlined in the chapter on Chronic Intestinal Indigestion. Peptonized milk is a valuable food in these cases.

Third.—Improve the blood state and general tone of the infant by keeping it out of doors as much as possible. Fresh air is very important, and if the weather conditions will not permit the fresh-air treatment at home, a change of climate may be recommended.

Fourth.—Treat the anemic condition directly by giving some form of organic iron, preferably combined with one of the malt preparations. The use of iron, however, in this condition must not be persisted in if the gastrointestinal canal is disturbed by it. In such cases the subcutaneous use of neutral citrate of iron in ¾-grain doses is of value (Friedlander). Arsenic is of no value in this disease.

## CHAPTER LXIII

### CHLOROSIS

Chlorosis is a secondary anemia characterized by deficient hemogenesis, producing a marked decrease in hemoglobin without a corresponding reduction in red blood corpuscles. This results in a low color index and a more or less characteristic greenish-yellow color of the skin.

**Etiology.**—It occurs almost exclusively in young girls about the age of puberty, and for this reason it may be inferred that nervous influences incident to the development of the sexual organs have something to do with its production. It is frequently seen in brunettes. It is extremely rare in boys. The most important predisposing factors are believed to be improper food, a deficiency of food, fresh air and sunlight. It occurs most commonly among factory and shop girls who spend long hours under unfavorable hygienic conditions. Constipation and intestinal intoxication, as Andrew Clark believed, are perhaps important factors. True chlorosis probably does not occur in infancy and young childhood, although the blood picture presented by secondary anemia at this age is, as already noted, of the chlorotic type.

**Symptomatology.**—The GENERAL SYMPTOMS are very like those of well-marked secondary anemia. The skin in advanced cases of chlorosis, however, has a yellowish-green pallor unlike that of secondary anemia, and the neurotic disorders which are so marked a feature of the secondary anemias, are not so pronounced in chlorosis, although the patient may be nervous, irritable and even hysterical, but chlorosis occurs later in life, and for this reason the nervous system, which is better developed, is not so profoundly affected. Cases of chlorosis do not suffer such marked nutritional disturbances as are present in well-marked secondary anemias. Shortness of breath, rapid and irregular heart action, cardiac dilatation, hemic murmurs, epigastric pain, gastrointestinal disturbances, headache, acne and irregular fever may occur. Constipation is a common and an important symptom. In older girls dysmenorrhea or amenorrhea may be present.

The BLOOD PICTURE is very characteristic, and by it the diagnosis is made. The hemoglobin is greatly reduced; it may be as low as 20 or 30 per cent., while the red blood corpuscles may be but slightly diminished in number. In severe cases, however, they are also much reduced, but not in a corresponding degree with the hemoglobin, so that the color index of the blood may be so low that the color of the red corpuscles is but faintly discernible. These almost colorless corpuscles vary in size and shape. Normoblasts appear in small numbers in severe cases, and are not an unfavorable sign, since they indicate blood regeneration. Microcytes may be present, and megaloblasts are extremely rare. The leukocytes are normal in number, but there may be a relative lymphocytosis.

**Diagnosis.**—The above blood picture occurring in girls between the ages of twelve and eighteen easily suffices to make a diagnosis.

**Prognosis.**—In uncomplicated cases the disease yields readily to treatment.

**Treatment.**—Fresh air, sunshine, and good food greatly facilitate the cure. A carefully selected diet of nutritious food, within the range of the digestive capacity of the patient, is important. The gastrointestinal canal must be kept in good condition, and it is very necessary that constipation be overcome with non-irritating laxatives. The proper use of iron is by far the most important therapeutic measure in the treatment. Under

its use the anemia gradually disappears, and the blood returns to its normal condition. Iron is perhaps best given in the form of Blaud's pills, 5 to 10 grains after meals, until the blood condition is materially improved; the dose may then be diminished to three or four pills a day. This treatment is to be continued for six weeks, or longer, if necessary. Saccharated carbonate of iron, reduced iron, and organic iron, in combination with malt preparations, may be used if gastrointestinal conditions demand them. Two or three grains of reduced iron, one or two grains of quinin and one-thirtieth of a grain of arsenic may be advantageously combined in the same capsule. Forchheimer recommends that five grains of betanaphthol or salol be given before meals, in connection with the iron therapy.

Patients recovering from chlorosis should not be allowed to return to the same unhygienic surroundings under which they developed the disease; otherwise relapses may occur.

## CHAPTER LXIV

### PERNICIOUS ANEMIA

This is a very grave form of anemia. It is characterized by a well-defined blood picture and by severe constitutional symptoms, which, although subject to strange remissions in severity, gradually grow worse until in the great majority of cases death results. It is an extremely rare disease in childhood, but when it does occur it presents the same clinical picture as in the adult.

**Etiology.**—The fish tapeworm, the hookworm and the malarial parasite may produce a form of pernicious anemia in which the blood picture is identical with the ordinary form of this disease, whose etiology is not understood. In childhood it is believed to be associated with syphilis, rickets, and chronic intestinal disorders, and rarely to be developed from severe forms of secondary anemia.

**Symptomatology.**—General Symptoms.—The symptoms are the same as in the adult. There is marked and progressive pallor of the skin, associated with muscular weakness, dyspnea, and heart symptoms similar to those found in severe secondary anemias. Vertigo, tinnitus, edema, gastrointestinal disorders, associated with pain, more or less marked disturbance of the functions of the spinal cord, and hemorrhages into the skin and from the mucous membranes very frequently occur. The diagnosis, however, of the disease is made from the blood picture.

Blood Picture.—There is a marked reduction in the number of red blood corpuscles, in advanced cases as low as 1,000,000 per c. mm., and even less. The hemoglobin is also reduced, but, as a rule, not to so great an extent as the corpuscles, so that each red cell contains an excess of hemoglobin. This produces a high color index, and is one of the most characteristic of the blood findings, especially in children, since the color

index with them in all other forms of anemia is comparatively low. Extreme poikilocytosis and basophilia are commonly present, and the size of the red blood corpuscles is in the average increased; megalocytes are common; this is an important diagnostic sign. Polychromatophilia frequently occurs. Normoblasts and megaloblasts are seen, the former in larger numbers. The red cells lose their rouleaux formation. The leukocytes are normal or subnormal in number, but the polynuclear cells are relatively diminished and myelocytes may be present.

Remissions.—This is a disease in which strange remissions may appear in the general symptoms and in the blood picture. During one of these remissions the patient improves over a period of some months, and upon this change false hopes are not infrequently founded. Suddenly, without apparent cause, there is an exacerbation in all of the symptoms, and the disease progresses steadily downward.

**Diagnosis.**—When a case presents the symptoms and blood picture of pernicious anemia, the stools must be carefully examined for evidences of the fish tapeworm and the hookworm, and the blood examined for the malarial parasite. These etiological factors excluded, the diagnosis of the ordinary form of pernicious anemia is made. In children, however, as previously noted, the blood picture in severe secondary anemias may closely resemble that of pernicious anemia, but the rarity of pernicious anemia in childhood and the comparatively low color index in secondary anemias, when taken in connection with the blood pictures of the two conditions, should make the diagnosis clear. If doubt remains for a time, the different course of the two conditions will clear the diagnosis. Eosinophilia in connection with the typical blood picture of pernicious anemia suggests intestinal parasites as the cause.

**Treatment.**—The treatment is the same as in the adult, and is unsatisfactory. It is questionable whether drugs of any kind influence the course of this disease. Arsenic, however, has been for a long time and is still in favor. It is to be given in gradually increasing doses until the physiological effects of the drug are produced, and then diminished to a moderate-sized tonic dose suitable to the age of the child. Careful attention to the gastrointestinal canal, looking to the avoidance of intestinal intoxication, is, perhaps, the most important therapeutic measure. Salol and betanaphthol should be given over a long period of time. A wholesome outdoor life is important.

In those cases due to the fish tapeworm, the hookworm or the malarial parasite, treatment should, of course, be directed toward the removal of these causative factors.

# CHAPTER LXV

## LEUKEMIA

Leukemia is a blood disease manifesting itself by a great increase in the leukocytes and by pathological changes in the bone marrow, spleen, liver and lymph nodes. It is very uncommon in infancy and childhood, but does occur.

**Etiology.**—The etiology of this condition is not at all clear, but in childhood it is believed to be etiologically related to syphilis, rickets, chronic gastrointestinal conditions, malaria, and severe secondary anemias.

**Symptomatology.**—The symptoms of this disease in childhood, as in adult life, appear in two rather well-marked clinical types, which are largely distinguished by the blood picture presented. The myeloid type is much the most common in the adult, and the lymphoid type comprises the majority of the cases in infancy and early childhood. In children these two types are very commonly mixed, the blood picture being a combination of the two. In the adult the types are more clearly defined.

Blood Picture.—In both types, hemoglobin and red blood corpuscles are diminished and nucleated forms are seen, but these changes are unimportant from the standpoint of diagnosis. It is the leukocyte blood picture which not only differentiates the two types, but distinguishes this disease from all others.

In the myeloid form we have an enormous increase in leukocytes; 100,000 to 500,000 are commonly found. A large number of myelocytes of different sizes are present; this is the most characteristic feature of the blood picture. The large mononuclears and the polynuclear and mononuclear eosinophiles are increased. The polynuclear neutrophiles and mast cells are also increased, but not to the same extent as the other cells mentioned. The special feature of this blood picture is an enormous leukocytosis with a great increase in myelocytes, polynuclear eosinophiles and mast cells.

In the lymphoid form the lymphocytes are greatly increased in number, even as high as 95 per cent. of the leukocytes present; myelocytes and mast cells may be present. There is a diminution in the amount of hemoglobin and red blood corpuscles, and some nucleated forms may be seen.

General Symptoms.—The onset and course of the disease are much more rapid in young children than in the adult. It commonly progresses to a fatal termination within a few months. The whole course of the disease may be embraced within a single month. The pallor of the skin and mucous membranes is very pronounced; gastrointestinal disturbances are common; vomiting is a frequent symptom; hemorrhages may occur from the nose and other mucous membranes; blood may be present in the

stools, and subcutaneous hemorrhages producing dark blue spots may occur from slight blows upon the skin. The appetite is lost, the child is nervous, irritable, and rapidly and progressively fails in strength. The spleen is always enlarged and may be enormously so, extending even to the crest of the ilium. The liver dullness is frequently greatly increased and the lymph nodes and other lymphoid structures, including the tonsils and adenoids, are also notably increased in size. As the disease progresses the child's strength rapidly fails, there is shortness of breath, the heart is weak, and rapid and repeated hemorrhages may greatly aggravate the prostration. The temperature may be normal, or may range from time to time as high as 102° or 103°F. Death occurs from exhaustion.

**Prognosis.**—It is questionable whether any of these cases occurring in infancy and young childhood ever recover.

**Treatment.**—The treatment is unsatisfactory, purely symptomatic, and directed toward the relief of suffering; any therapeutic measures to this end are justifiable. The X-ray treatment of these cases, after a thorough trial, has been found to be without therapeutic value.

## CHAPTER LXVI

### PURPURA

Purpura is the name applied to a condition in which there is a transitory and nonhereditary hemorrhagic diathesis, caused by toxins and other contributing etiological factors. It is characterized by a skin eruption produced by small spontaneous hemorrhages occurring in the subcutaneous tissues. The hemorrhagic spots thus produced are bluish, but gradually fade to a brown and then yellow color. This yellow pigment is slowly absorbed, removing all discoloration, so that the skin may present a normal color within a week or ten days after the initial lesion. New crops of purpuric spots occurring from time to time may prolong the eruption for an indefinite period. These purpuric spots do not disappear upon pressure. The eruption may appear as fine petechial hemorrhagic points, but, as a rule, the spots are larger, approaching in size a split pea, and sometimes large, irregular hemorrhagic patches occur, resembling an ordinary bruise of the skin. It may occur on any part of the body, but it is most common, and usually first appears, on the legs, especially over the shin bones. The frequent association of purpura with urticaria, erythema exudativum, and localized edema, in the various symptom groups classified as "purpuras," indicates that the same toxins may produce these various skin lesions by their action through the nervous mechanism that controls blood and lymph vessels. This association of symptoms in part helps to establish the more or less clearly defined symptom groups below referred to. The purpuric eruption very commonly occurs as a symptom of constitutional disorders, toxic, cachectic and nervous in their character.

These symptomatic purpuras are not separate clinical syndromes, the rash being but one of a large group of symptoms which belong to some well-defined constitutional disorder. In other instances the purpura occurs as the all-important clinical feature of a distinct syndrome, whose etiology is more or less obscure. These cases are spoken of as idiopathic, to distinguish them from the clearly defined secondary purpuras. It may be that the various forms of purpura are merely clinical syndromes representing different phases and different degrees of the same hemorrhagic diathesis, but they are here described as distinct disease pictures in order that the clinical course of the various symptom groups of this condition may be more clearly understood.

The symptom-complex of the following idiopathic purpuras will be here briefly described: Purpura simplex, purpura fulminans, purpura hemorrhagica, Henoch's purpura, and purpura rheumatica.

**Etiology.**—It is believed that nearly all of the worst forms of purpura are due to toxic substances circulating in the blood, and that they act not only upon the nervous system, producing profound vasomotor disturbances, but also directly on the endothelial lining of the smaller blood vessels, producing degenerative changes. It is known that blood coagulation is impaired, that the blood platelets are diminished and that the blood clot formed from purpuric blood does not contract firmly. It is believed, especially by French writers, that functional disturbances of the liver are etiologically related to this condition.

**Symptomatic Purpura.**—Symptomatic purpura occurs not uncommonly in association with nervous disorders, but this form is rare in children. It may also rarely result from the administration of drugs, such as iodid of potash, mercury, belladonna, antipyrin, quinin, salicylic acid, chloral, ergot, and potassium chlorate. A very pronounced form of purpura can be quickly produced by snake venom. The most common cause of secondary purpura in infancy and childhood are the acute infections. It may occur as part of the symptom-complex in smallpox, diphtheria, scarlet fever, measles, cerebrospinal meningitis, septicemia and septic endocarditis, and when it does occur in these conditions it adds great gravity to the prognosis. In infancy, secondary purpura may occur in hereditary syphilis, rickets, tuberculosis, chronic ileocolitis, the severe secondary anemias, leukemia, and in all conditions producing a profound malnutrition.

**Purpura Simplex.**—This condition is characterized by a purpuric eruption usually symmetrical in its distribution. It first appears on the legs and may be confined to the lower extremities, but it usually appears in other parts of the body. The rash, commonly petechial in character, appears suddenly without constitutional symptoms. There is little or no fever. Nausea, vomiting and digestive disturbances may or may not be present. The disease runs its course, as a rule, to a favorable termination within from two to four weeks. The prolongation of this condition is due to the fact that the eruption comes out in successive crops.

**Purpura Fulminans.**—This is a rare and very severe form, commonly

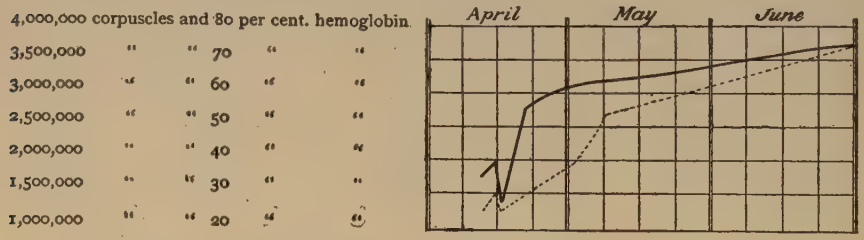
terminating fatally within one or two days; death may occur within the first twelve hours, or may be postponed for four or five days. In a case which I reported some years ago death occurred within twenty-four hours. This case was a helpless, idiotic, epileptic child, nine years of age, living under very unfavorable hygienic surroundings, and in the same room with two cases of malignant diphtheria, one of which died on the same day as this patient. There was no fever and no hemorrhage from mucous membranes; the onset of the disease was marked by irritability, refusal to take food, and the appearance of a great number of dark blue hemorrhagic spots over the legs, scrotum, and abdomen, these spots rapidly increasing in size until they ran together, producing a dark blue discoloration over the parts above named. Small hemorrhagic spots appeared also over other portions of the body, profound prostration was quickly followed by delirium, stupor, coma, and death. This is a typical clinical picture of this symptom group. Convulsions, vomiting and high fever may be present in these cases, and albumin is usually found in the urine. In the case above detailed the inference is that the disease was produced by the toxins of diphtheria, although no evidence of diphtheria appeared in the throat. Many of these cases are believed to be foudroyant cases of smallpox, measles, scarlet fever and other infections. The patients, however, as a rule, do not live long enough to develop the characteristic symptoms of these diseases.

**Purpura Hemorrhagica.**—Purpura hemorrhagica is characterized by hemorrhages from mucous membranes, and is thereby distinguished from other forms of purpura. These hemorrhages, which are the characteristic feature of the disease, occur apparently without exciting cause. Their most common site is the nose; bleeding may also occur from the mouth, especially the gums, from the kidneys, the intestinal canal, and, in fact, from any mucous membrane. In a case which I reported, the bleeding from the nose persisted almost continuously for six days, and bled at intervals during the next three or four days, until on the tenth day of the disease a blood examination showed 21 per cent. of hemoglobin and 1,300,000 red blood corpuscles. The resulting anemia which occurs in some of these cases is very great, and in those cases that recover, the increase in the number of corpuscles is so much more rapid than the increase in hemoglobin, that the anemia assumes for a time a chlorotic type. This is illustrated in the chart taken from the report of the case above referred to.

The purpuric rash in this condition is widely distributed over the body, beginning, as a rule, on the legs; the hemorrhagic spots vary in size from a pinhead to a silver dollar. In the case above referred to, the baby sister of the patient struck him a slight blow on the forehead with an Easter egg, producing a dark blue, irregular hemorrhagic spot. The spots, however, as a rule, appear spontaneously and may occur in crops from time to time throughout the course of the disease. Fever, nausea, vomiting and gastrointestinal disturbance may or may not be present. The temperature, in the early stages, ranges in the neighborhood of 102°F. Albumin

may be found in the urine, and acute nephritis is the most common serious complication. Arthritis may occur, but is not, as a rule, a feature of this disease. Edema may also be present, but erythema and urticaria, so commonly present in other forms of purpura, are rarely seen in this condition.

The disease is a serious one, lasting from two to eight weeks, but most of the cases recover. In rare instances the disease may resemble typhoid fever, having a continuous fever, intestinal hemorrhages, great prostration, delirium, and, in fatal cases, coma. These cases, however, should be easily differentiated from typhoid by the absence of the Widal reaction, and by a careful study of the two symptom groups.




Solid line = number of corpuscles. Broken line = percentage hemoglobin.

FIG. 82. DIAGRAM SHOWING BLOOD CHANGES IN A CASE OF PURPURA HEMORRHAGICA.

**Henoch's Purpura.**—This form, first described by Henoch, occurs most commonly in children. The purpura is associated with attacks of severe abdominal pain and polyarthritis. The onset may be marked by headache, fever, prostration, and the outbreak of a purpuric rash. In some cases, however, the arthritis or intestinal colic may precede the eruption, which is frequently accompanied by urticaria, erythema, and angioneurotic edema. Osler has called attention to the fact that in this form of purpura, as in others associated with erythema, there may be great variations in the appearance of the skin; the purpura, erythema, urticaria and localized edema may all be present in an individual case, or again various combinations of these skin lesions may occur, and any one may be absent. Recurring attacks of severe abdominal pain is the characteristic symptom. It may be associated with violent and prolonged vomiting, and early constipation is followed by diarrhea; the stools may contain blood. The pain is usually referred to the umbilicus, and is associated with abdominal resistance and with tenderness over the upper portion of the abdomen. Arthritis occurs in practically all of the severe cases. There may be pain and swelling in one or more joints, and successive joints may be involved.

The acute symptoms of an attack last from a few days to a week; within this time the eruption fades, the pain and tenderness of the joints disappear, the gastrointestinal disturbances cease, and the patient is apparently convalescent. Within a week, however, the whole symptom group above detailed may recur, and repeated attacks of this character with intervening periods of apparent convalescence may prolong the illness for months, but true convalescence is established in the majority of cases

within a month or six weeks. In a few instances the disease becomes chronic and lasts over a period of years. Acute nephritis is a dangerous, and by far the most common, complication; cerebral hemorrhage, endocarditis and pericarditis may occur.

The prognosis, on the whole, is good, especially in children. About ten per cent. of these cases die from various complications.

**Purpura Rheumatica** (*Schönlein's Disease*).—This is seen in older children, but more commonly in young adults, and is not in any way etiologically related to rheumatism, although it may begin with pharyngitis and tonsillitis. The purpuric rash resembles that seen in simple purpura. It consists largely of petechiæ associated with slightly larger ecchymotic spots, distributed chiefly over the lower extremities, but other parts of the body may be involved. In some cases the eruption is more marked over the swollen joints and spreads from joint to joint with the arthritis.

The chief characteristic is the multiple arthritis, and all cases of arthritis associated with simple purpura, uncomplicated by abdominal colic and by hemorrhage from mucous membranes, are classed under this syndrome. The arthritis usually affects the knees and ankles in these cases and commonly disappears within a week.

When the arthritis is the initial lesion and is associated with fever, as it is in many of the cases, a diagnosis of rheumatism is ordinarily made. The character of the trouble, however, is soon made clear by the appearance within a few days of the purpuric eruption and possibly the subsequent association of erythema, urticaria and localized edema with the purpuric rash. The erythema, urticaria, edema of the hands and feet and even typical angioneurotic edema, which is associated with the purpura in many of the cases of purpura rheumatica, present a clinical picture which may closely resemble Henoch's purpura, with the exception of the gastrointestinal symptoms of that disease. Albuminuria may occur, but nephritis and other complications seen in Henoch's purpura are very rare.

These cases run a benign course and are commonly convalescent within a month; relapses, however, are not uncommon.

**Treatment.**—The treatment of symptomatic purpura calls, in the first place, for the treatment of the underlying causative condition, and, in the second place, for the routine treatment of purpura as here outlined, if the treatment recommended is not contraindicated by the primary disease.

General Treatment.—General treatment applicable to all forms of purpura. Absolute rest in bed until all symptoms are under control is of great importance. The patient should be kept quiet, and should be in the hands of competent nurses who can protect him from all possible injuries to the skin and mucous membranes. Fresh air, day and night, is essential. When the weather will permit and the surroundings are favorable, the bed of the patient should be placed out of doors for as much of the twenty-four hours as is practicable.

Diet.—Milk and cereals should be the basis of the diet, and orange juice and other fruit juices should be given. Since purpura is a toxic

condition, and since the kidneys very commonly give way under the irritation of excreting these toxins, it is most important that these patients should be dieted as they are in scarlet fever with the idea not only of eliminating toxins, but of protecting the kidneys and other excretory organs. Rich, albuminous foods, and strong beef broths, should, therefore, be avoided in the treatment of all forms of purpura during the acute stage of the disease. In addition to the diet above recommended, water should be given freely to assist in the elimination of toxins and in the flushing out of the excretory organs. During convalescence the diet may be increased and fruits, vegetables and albuminous foods may be given.

Cathartics serve a useful purpose and are indicated in all forms of purpura not complicated by intestinal hemorrhage. Calomel, Rochelle salts, sulphate of soda, sulphate of magnesia and phosphate of soda not only remove offensive matter from the intestinal canal and eliminate toxins through the intestinal wall, but they also unload the portal circulation and perhaps favorably influence the functional inactivity of the liver which is believed to be present in most cases of purpura.

Hydrotherapy.—Elimination of toxins should also be promoted through the skin. This may be accomplished by the giving of one or two warm alkaline baths each day; common salt, sea salt or bicarbonate of soda may be used in these baths, and great care must be taken in handling the patient, that fresh ecchymoses may not be produced by bruising the skin.

Medical Treatment.—Calcium lactate is believed to exert a favorable influence by increasing the coagulability of the blood; it may be given in five-grain doses to a child six years of age, increasing the dose one grain for each year of life until the maximum dose of 15 grains three times a day is reached. Fowler's solution has been extensively used and is believed to be of value in these cases; it should be given, as in chorea, in gradually increasing doses until improvement begins, or until the patient is taking 10 drops three times a day. It is perhaps of special value in those cases that are believed to be of cachectic or neurotic origin, and are associated with marked nervous symptoms. It is contraindicated in Henoch's purpura and in all forms where there is gastrointestinal irritation.

Iron is a remedy of little or no value during the acute stages of this disease, but is a remedy of very great value during convalescence. Its special value is in those cases where a more or less marked anemia results. The preparation of iron selected will depend largely upon the age of the patient and the condition of the gastrointestinal canal. In younger children the organic iron preparations and the saccharated carbonate given with some form of malt are most valuable. In older patients Blaud's pills may be given.

Treatment of the Special Forms.—The prime object in the treatment of *purpura hemorrhagica* is to control as soon as possible the bleeding from the mucous membrane. Where the bleeding surface can be reached, as in the mouth or nose, the parts should be irrigated with a 1 to

1,000 adrenalin solution, or should be packed with cotton saturated with this solution. In hemorrhages from the stomach adrenalin may be taken internally in the hope that it may act locally upon the bleeding vessels. In the control of hemorrhage the bleeding part should be elevated and absolute rest insisted on; the patient should not be allowed to do anything for himself that can be done by others. If there be hemorrhage from the stomach or intestine, the prolonged application of ice to the abdomen should be resorted to, and where these measures fail, 5 or 10 minims of adrenalin solution may be injected hypodermically. The hemorrhage once being controlled, rest in bed and absolute quiet should still be insisted upon for a period of a week or ten days, and during this time the general treatment of purpura as above outlined should be carried out. During convalescence the profound anemia which has resulted from the hemorrhage demands the use of iron in large doses. Later the iron may be combined with arsenic, and this treatment continued until all traces of the anemia have disappeared.

During the acute stage of *Henoch's purpura* the gastrointestinal condition precludes giving anything except the lightest food, such as cereal waters, to which milk may be added. Laxative medication, preferably castor oil or sulphate of magnesia, is indicated to relieve the abdominal pain. Osler believes that the pain in this condition is due to an edematous condition of the intestinal wall. This may be the explanation for the relief from intestinal pain which the saline cathartics afford in this disease. In some instances it may be necessary to give morphin hypodermically. Ice-bags to the abdomen may also be of benefit. As the intestinal symptoms come under control the bowels are still to be kept open with saline cathartics, and milk and cereal gruels are to be continued until all danger from nephritis is passed. The general treatment is the same as that above outlined.

The other forms of purpura require no special treatment other than that above given. In *purpura fulminans* all treatment is unavailing. In *purpura rheumatica* the disease runs a benign course and responds to the routine treatment of purpura. Salicylates are of no value in this disease.

## CHAPTER LXVII

### HEMOPHILIA

Hemophilia is a rare disease, characterized by an hereditary and long-continued, if not permanent, predisposition to severe and ofttimes uncontrollable hemorrhages, which may be precipitated by traumas so slight as to be undiscoverable. Patients suffering from this disease are popularly known as "bleeders."

**Etiology.**—Comparatively little is known of the pathology and etiology of this condition. It is, however, distinctly hereditary, running through families for many generations. In some of the families studied the disease

has persisted for two hundred years. The hereditary tendency is, in the vast majority of instances, transmitted through females, themselves non-bleeders, to the male members of a family, thus skipping a generation. Direct transmission from parent to child is very unusual. It has a great predilection for males; females are comparatively rarely affected, the proportion being about as one to twelve. It is not unusual in hemophiliac families to find more than half of the males descended from the female members of the family suffering from this disease, while none of the females and none of the males descended from male members of the family have the disease. In rare instances, however, it should be remembered that the disease may descend through the male line and that the females may be affected.

*Age.*—In the new-born hemorrhages from this cause are rare, but do occur. This disease, however, nearly always begins in early childhood. In 65 per cent. of the cases the first hemorrhage appears during the first or second year of life, and children of hemophiliac families who have shown no symptoms before the tenth year of life are comparatively safe. In rare instances, however, it may appear in adult life. It is especially rare in warm climates.

**Symptomatology.**—The characteristic hemorrhage is not, as a rule, violent, but it is long-continued and, if controlled, sooner or later commonly recurs. The surface from which the blood oozes usually shows no sign of trauma. The first hemorrhage is rarely fatal, but the subsequent ones usually cause death during childhood; less than 15 per cent. reach maturity. These uncontrollable hemorrhages may last for weeks, or until the child dies from exhaustion. Spontaneous hemorrhages may be preceded by restlessness, nervousness, vertigo, circulatory disturbances, and other prodromal symptoms. If the patient lives to maturity, the tendency to these hemorrhages gradually grows less, so that he may even outgrow his hemorrhagic predisposition. Very rarely, dangerous and even fatal hemorrhages occur after middle life.

A family history of hemophilia, which is present in nearly every case, is hardly less important in making the diagnosis than the characteristic hemorrhages above described, since with this family history the diagnosis may be made with the onset of the first hemorrhage, and without it one must wait until repeated severe and almost uncontrollable hemorrhages have occurred before making the diagnosis.

In two of the three cases[1] reported by me, the bleeding point was in the median line of the upper surface of the tongue. Hemorrhages most commonly occur from mucous membranes, especially of the nose and throat. Catarrhal diseases of the nose and throat, and diseases of the gums, may precipitate serious and even fatal hemorrhages. Bleeding may also occur from the bowels, the stomach, any portion of the skin, and, in fact, from any part or into any organ of the body. Subcutaneous hemorrhages producing hematomas are common. These hematomas may be very large,

[1] *Medical News*, 1892.

and may be associated with fever and gastrointestinal symptoms. Hemorrhages into the skin may produce ecchymotic spots, which may lead to some confusion in the diagnosis of this disease from purpura. The ecchymoses, however, in hemophilia are large, irregular in shape, and not, as a rule, widely distributed.

Arthritis occurs, sooner or later, in almost every case. The knees and elbows are most commonly involved, but the smaller joints may also be affected. The swelling occurs rapidly and is produced by hemorrhage into the joint from the synovial membrane. In the beginning there is no tenderness, redness, or inflammation, although pain may be present as a result of the tension. The inflammatory stage quickly follows, during which the joint is somewhat tender and red, showing a subacute inflammation. From this injury the joint may entirely recover, or permanent ankylosis may result. Fever and pain may accompany the joint symptoms, but they are not so pronounced, nor so transient in character, as they are in the arthritis associated with purpura.

*Pronounced anemia* and great exhaustion follow the long-continued hemorrhages that occur in these cases. The microscopic blood picture is that of severe secondary anemia. The pulse becomes rapid and thready, the patient is nervous, faint, and complains of shortness of breath and great exhaustion. If the hemorrhage is controlled the patient slowly convalesces, perhaps to suffer from another attack, to which he succumbs from exhaustion.

**Diagnosis.**—In the vast majority of instances the family history, the repeated hemorrhages, and the subacute arthritis make the diagnosis plain. In rare instances, however, the occurrence of a purpuric rash with these symptoms, and the absence of a family history of hemophilia, may make the differential diagnosis between this condition and purpura hemorrhagica a very difficult matter.

**Prophylaxis.**—The members of hemophiliac families should be advised against marriage. This applies especially to the females, since it is through them the disease is most likely to be transmitted. The children of these families should, during infancy and young childhood, be most carefully guarded from injury, and should, if possible, live in a warm climate, since mild, semitropical climates sometimes exercise a decidedly protective influence against the hemorrhages of this disease; it is possible that the beneficial results of a warm climate are due to the comparative freedom which such a climate offers from catarrhal diseases of the nose and throat. As a prophylactic measure the gums and mucous membranes of the mouth should be carefully washed every day with a simple alkaline antiseptic, and all surgical measures of every kind, including the pulling of teeth and circumcision, should be carefully avoided.

**Treatment.**—In the treatment of an attack the control of the hemorrhage demands first consideration. Long-continued compression of the bleeding part is the most valuable measure. Adrenalin, a sterile solution of gelatin, and perchlorid of iron may be used with compression. The

great difficulty in most instances is to reach the bleeding point in such a way that these measures may be satisfactorily employed. In one of the cases reported by me, the tongue was compressed by a clamp, which held a small piece of cotton, saturated with Monsell's solution, against the bleeding point on its anterior surface. The grasp of the instrument was now and then slightly relaxed to allow better circulation in the tip of the tongue. After two hours of such treatment the clamp was removed and a hemorrhage which had lasted for five consecutive days was controlled.

Calcium lactate in 5- to 15-grain doses, depending upon the age of the child, should be given three times a day for a period of four or five days, or until the hemorrhage is controlled. A number of observers have reported good results from this remedy, and have recommended that it be given at least one day in every week in the subsequent treatment of these cases.

Serum Treatment.—Recent reports indicate that standardized animal blood serum is a valuable remedy in stopping the hemorrhage and in controlling, at least temporarily, the hemorrhagic tendency. It is to be injected in doses of 10 to 20 c. c., and this dose repeated, if necessary, every second day until 100 c. c. have been given. The local application of the serum to bleeding mucous membranes is also recommended for controlling hemorrhages. If a simple standardized sheep or other animal serum is not available, one may use antistreptococcic serum. Ten c. c. of normal human serum, when it can be had, is a safer and more efficacious remedy. Direct transfusion of human blood has been successfully used in the treatment of the hemorrhagic diseases of infancy and childhood. These remedies may not permanently remove the cause of the disease, although they produce great temporary benefit by restoring the impaired blood coagulability.

## CHAPTER LXVIII

### HODGKIN'S DISEASE

Hodgkin's disease, also described under the synonyms *adenie, lymphadenoma* and *pseudoleukemia,* is characterized by a progressive, painless enlargement of lymph nodes, usually beginning in the neck, associated with a progressive anemia and frequently with the formation of nodules in the spleen and other internal organs.

**Etiology.**—Nothing is known of the causes of this disease. Tuberculosis is not infrequently associated with it as a secondary infection. It occurs most commonly in childhood and early adult life, is not infrequent in the second and third decades, but is comparatively rare after forty.

**Pathology.**—A symptom-complex closely resembling Hodgkin's disease may be presented by other pathological processes, such as lymphosarcoma,

but the term should be confined to a chronic inflammatory process which produces an enormous enlargement of lymph nodes, and results in the deposit of lymphatic nodules in the spleen, liver, subcutaneous tissues, and other parts of the body. The exciting cause of this progressive chronic inflammatory process is not known, but the microscopical changes produced are the same as in other chronic inflammations.

**Symptomatology.**—General Symptoms.—Enlargement of lymph nodes beginning, as a rule, in the posterior triangle of the neck, is the characteristic symptom. It may rarely begin in the inguinal, axillary, mediastinal, or other lymph glands. The enlargement of these nodes produces painless tumor masses which spread by continuity to neighboring glands on the same or opposite sides of the neck or into the mediastinal or axillary regions, in this way producing enormous tumor masses, which are firm, hard and nodular, but are freely movable, and show no tendency to suppuration. The disease may be uniformly progressive, or there may be periods of apparent quiescence followed by periods of rapid growth. As it progresses, pressure symptoms may result from these large tumors, impinging on the trachea, bronchi, blood vessels, nerves, bile ducts, ureters and other tissues. There is progressive anemia, weakness, and cachexia. The gradually increasing dyspnea, which may end in strangulation, is the most terrible of all these pressure symptoms.

The spleen is moderately enlarged in a majority of the cases, and the liver may also be increased in size. Nodular masses in the subcutaneous tissues may occur.

The fever is very variable; afebrile cases may occur; as a rule, there is a low continued fever, rarely rising above 103°F., which continues for months; in other instances it may be intermittent, and in those cases, with periods of apparent quiescence, there may be periods in which the temperature is normal, followed by periods of severe pyrexia, during which there is a rapid progression of the disease.

Blood Changes.—The only important blood change which occurs in this condition is a simple secondary anemia, caused by an almost equal reduction in hemoglobin and red blood corpuscles. This anemia is progressive, and as the disease advances becomes extreme. In the last stages there may be less than 30 per cent. of hemoglobin and less than 2,000,000 red blood corpuscles per c. mm. The leukocyte picture is practically normal; there may, however, be a slight leukocytosis of 15,000 or 20,000.

**Diagnosis.**—In lymphosarcoma the disease does not confine itself to the lymphatic glands, so that the tumor mass is softer and the nodules less clearly defined than in Hodgkin's disease. When in doubt, a microscopical examination of one of the superficial lymph nodes removed from the mass will definitely determine the character of the growth.

Tuberculosis may rarely produce large, painless lymphatic tumors of the neck, which may grow in size and extend from node to node over a period of months, without showing any tendency to break down. These

cases may be confused with Hodgkin's disease, but, as a rule, the diagnosis can be made by the tuberculin skin reaction, and by other symptoms of lymph-node tuberculosis. The examination of a superficial lymph node removed from the mass will establish the diagnosis.

From leukemia and secondary anemias with splenic enlargement this disease may be differentiated by the blood examination. The so-called pseudoleukemia of infancy bears no resemblance whatever to this disease.

**Prognosis.**—The disease is probably always fatal, usually terminating within two years, but patients may live for four or even five years. Death may occur from tuberculosis, secondary anemia, septic or other infections, or from the pressure of the tumor masses on vital structures.

**Treatment.**—There is no treatment that exerts any curative influence. The removal of the tumor masses by surgical measures may give temporary relief from pressure symptoms, but has no influence on the progress of the disease. Arsenic has been in general use for a long time in the treatment of this condition. It should be given in large doses over long periods of time, and the consensus of opinion is that it sometimes diminishes the size of the tumors, and frequently causes a temporary cessation in their growth. It exerts no curative action, and probably has little influence in prolonging life.

The X-ray, at the present time, is the remedy most in vogue in the treatment of this condition. Under its use the large lymphoid tumors may diminish in size, and in some cases almost disappear. When the treatment is discontinued, however, the tumor masses reappear. This treatment, therefore, has no curative influence. It is believed, however, that if the X-ray is used to reduce the size of these lymphoid masses when they become large or exert uncomfortable or dangerous pressure symptoms, the life of the patient may be made more comfortable, and may, perhaps, be prolonged.

## CHAPTER LXIX

### SIMPLE ADENITIS

Simple adenitis is an inflammation of lymph nodes not produced by tuberculosis or syphilis. It is not a distinct disease, but a secondary condition due to infection with pathogenic microorganisms, commonly the pyogenic cocci, which have found their way from nearby inflammatory processes in direct communication with the infected nodes. The pyogenic infection thus produced results in an acute inflammation and general hyperplasia of the gland tissue, which may subside without suppuration, but which, especially in infancy, very commonly results in the breaking down of the lymph nodes, and more or less involvement of the surrounding cellular tissue, with the final discharge of the abscess through the skin or into some cavity of the body.

The deep cervical lymph nodes are by far the most commonly affected, since they are in direct communication with the mucous membranes of the pharynx, nose, throat and mouth, so commonly the sites of disease in the child. These glands become affected in tonsillitis, pharyngitis, inflammation of the adenoids and rhinitis. The submaxillary glands are enlarged in stomatitis and ulcerated teeth.

The superficial cervical lymph nodes are commonly enlarged from eczema of the scalp and face, or from infected wounds of these regions.

Simple adenitis of other lymph nodes of the body is less common. The inguinal nodes may be affected from vulvovaginitis in infancy and from other local inflammatory processes in that region. The axillary lymph nodes are commonly inflamed as the result of vaccination, and may be enlarged from other infected wounds of the upper portion of the arms. Adenitis of the bronchial lymph nodes is associated with disease of the lungs and smaller bronchi, and the deep-seated abdominal lymph nodes may become affected in gastroenteritis and other diseases of the enteric canal. Certain of the acute infections, especially influenza, scarlet fever, diphtheria, and measles, are almost always associated with more or less enlargement of lymph nodes. The adenitis, however, which is produced by the acute infections and by disease of the lungs and gastroenteric canal, as well as that which results from tuberculosis and syphilis, is discussed in connection with these diseases. It only remains, therefore, for us to call attention very briefly here to the fact that simple adenitis, especially of the cervical glands and more rarely of the inguinal and axillary glands, occurs in young children, as the result of some simple nearby inflammatory process, and that the adenitis in these cases may be so severe that the causative condition may almost be lost sight of in the symptom group that follows.

Age is a most important predisposing factor of simple adenitis. The great majority of the cases occur under two years of age. The younger the child the greater the probability that the resulting inflammation will end in suppuration.

**Symptomatology.**—The enlarged lymph nodes are readily seen and felt. They appear as hard, painful, tender masses, round or oblong in shape, in the subcutaneous tissues or beneath the superficial muscles. Neighboring nodes may be so agglutinated by the accompanying cellulitis that a large tumor mass may form, in which the individual nodes may be lost. Fever is present during the acute stage and lymphocytosis develops. As the inflammation proceeds the overlying skin becomes red, and if suppuration occurs, as it frequently does in infancy, a softening of the inflamed glands may be felt. The abscess points and finally breaks through the skin, and with the discharge of pus the inflammation quickly subsides and the wound heals. In the great majority of cases, and this is especially true in children over two years of age, the tender, tumor-like mass, large or small, after the second or third week gradually diminishes in size, loses its tenderness, and the individual nodes which were formerly

lost in the tumor mass may now be made out. In the majority of instances these glandular swellings gradually disappear, so that a return to normal conditions may be expected in from four to six weeks. If, however, the source of irritation which originally caused the lymph node enlargement continues, a simple chronic adenitis may result. In these cases the lymph nodes may remain enlarged for many months, but the symptoms are much less pronounced than in the acute form above described.

In simple bronchial and abdominal adenitis the diagnosis may be confirmed by radiographs and by the same physical signs, which have been detailed in the chapter on tuberculosis.

**Diagnosis.**—The diagnosis of syphilitic and tuberculous adenitis has been carefully considered elsewhere, and if these symptom groups are kept in mind there should be no difficulty in making a differential diagnosis of simple adenitis. It may be noted here, however, that the presence of a local exciting cause, such as disease of the mucous membranes of the nose, mouth or throat, vaccination or vulvovaginitis, may speak in favor of a simple adenitis, and also that, under two years of age, simple adenitis is much more common than tuberculous adenitis, and much less common after this period of life. The diagnosis, however, is usually made by excluding tuberculosis and syphilis by reason of the absence of the characteristic symptom-complexes of these diseases.

**Treatment.**—In every instance the cause of the adenitis should be sought and treated. Most of these cases are dependent upon diseases of the lymphoid tissues of the pharynx. The most important part of the treatment, in such cases, is the careful treatment of all catarrhal conditions of the nasopharynx. Inunctions of unguentum Credé and guaiacol [1] into the tissues surrounding the enlarged lymph nodes are of great value and should be resorted to in all severe cases. The technique of this treatment is given in the chapter on Scarlet Fever. Cold compresses are of value in many cases. When abscess formation can be definitely made out, an incision should be made and the pus evacuated. In subacute and chronic cases, where the glands remain large and tender with no tendency to suppuration, the application of flexible collodium, as recommended by Forchheimer, by exerting a steady pressure upon these glands, promotes their absorption. Iron and iodin tonics are of value. In older children the freshly prepared syrup of the iodid of iron is followed by good results. In all cases fresh air and properly selected food are almost of as much value as they are in the treatment of tuberculous adenitis.

[1] See Tuberculosis.

## CHAPTER LXX

### STATUS LYMPHATICUS

Status lymphaticus is characterized by a hyperplastic enlargement of the thymus gland and other lymphoid structures. This lymphatic hyperplasia is commonly associated with a lowered vitality, a chloranemia, and a well-marked dyspnea, which may be aggravated into pronounced asthmatic attacks or end in sudden death.

**Anatomy and Pathology.**—The thymus is a ductless gland composed of lymphoid tissues, holding remnants of the epithelial structures which in early life predominated in its makeup. It consists of two lobes joined together in the median line. It is situated behind the upper portion of the sternum occupying the superior strait of the anterior mediastinum. Below, it rests upon the pericardium and extends upward over the great vessels of the heart into the neck, resting on the trachea as far up as the thyroid gland. Laterally it is in contact with the vagi, the phrenic nerves, the innominate and common carotid arteries. Its association, however, with the trachea in the narrowest portion of the chest, between the manubrium sterni and the spinal column, is of special pathological importance, since enlargement of the gland in this narrow confined space must necessarily contract the lumen of the trachea. It is relatively large in infancy and early childhood, and slowly increases in size up to the fifteenth year, after which regressive changes slowly take place which diminish its size and physiological efficacy. The size of this gland varies greatly in different individuals of the same age. Warthin accepts 7 grams as the average weight in the new-born, and 15 grams as evidence of a hyperplastic condition of this organ. It is generally believed that the thymus furnishes an internal secretion which exerts an important influence on nutritional processes, and especially, as the writer believes, on the functional efficiency of all lymphoid tissues. Its period of greatest functional activity is during fetal life and early childhood. This function diminishes most rapidly after the fifteenth year, but probably remains more or less active throughout life. Svchla offered the theory that hyperthymization was the important cause of status lymphaticus.

The most important pathological condition in status lymphaticus is a true hyperplasia of lymphoid tissues, most marked in the thymus gland. This gland may be greatly enlarged, weighing forty or fifty grams, and, according to Warthin, there may also be a congestion or edema which further increases its size and leads to pressure upon the trachea and other important structures held in the narrow, closely confined and unyielding space beneath the upper part of the sternum. The enlargement of the spleen, tonsils, adenoids, lymphatic glands, and lymphoid tissue in general, so commonly associated with the enlarged thymus, is also hyperplastic in character. There is hypoplasia of the general arterial system;

marked blood changes occur and the heart muscle may be weak and dilated. Rachitic and syphilitic changes are present in some of the cases.

There is no doubt that enlargement of the thymus may by compressing the trachea produce dyspnea and violent attacks of asthma. This is proven by the fact that these symptoms may be relieved either by extirpating the enlarged thymus, or by inserting into the trachea beyond the point of constriction a long intubation tube. It does not follow, however, that these symptoms may not in other cases be produced by pressure on other tissues or by toxins, or by hyperthymization (Svehla).

**Symptomatology.**—The most important symptom and the one that usually calls attention to the condition is the *dyspnea,* which may vary in severity from a mild stridor to a violent attack of asthma terminating in the death of the infant. A severe spasmodic cough almost always accompanies this stridor. The cough may produce vomiting, be associated with cyanosis and greatly aggravate the dyspnea, precipitating at times severe asthmatic attacks.

*Thymic asthma* is an exaggeration of the thymic dyspnea or stridor. The first difficulty in breathing associated with an irritable or spasmodic cough usually makes its appearance in early infancy, sometimes soon after birth. These symptoms may disappear and reappear from time to time, perhaps gradually increasing in severity, over a period of months and even years, until the disease becomes so advanced that asthmatic attacks are precipitated by indigestion, influenza, slight catarrhal conditions in the nose or bronchial tubes, or by any pathological condition which irritates lymphoid tissues. In severe cases, throwing the head backward may produce a violent asthmatic attack. As the disease progresses these asthmatic attacks become more violent and recur without apparent cause, severe dyspnea being almost continuous. In these cases marked cyanosis and temporary suspension of breathing may threaten suffocation; sudden death may occur.

Sudden deaths in infancy from slight or unknown causes are commonly due to the status lymphaticus. Deaths from slight surgical operations, such as the removal of adenoids, and circumcision, and from slight injuries or from sudden shock, such as coming in contact with cold water, as in bathing, may be of this character. In some instances death may occur without apparent cause; the child may be playing about when suddenly it becomes cyanotic, slightly convulsed, and quickly dies of respiratory failure, or it may be found dead in bed. These latter cases no doubt include some of those thought to be due to "overlaying." The exact modus operandi of these deaths is not understood, and just what rôle hyperthymization and pressure on the trachea, the pharynx, or pneumogastrics play is yet to be decided. In perhaps most of these cases a diagnosis might have been made if the disease had been suspected and the patient subjected to a careful examination during life. It should also be remembered in this connection that sudden death in infants may occasionally occur from causes entirely apart from the status lymphaticus.

Laryngeal spasm and acute pulmonary congestion are among such causes.

Sudden deaths from anesthesia in infancy and childhood are almost always due to this cause, and the diagnosis in most of these cases is made on the post-mortem table, by the finding of an enlarged thymus, a weak and dilated heart muscle, and perhaps other signs of the status lymphaticus. Chloroform is considered more dangerous than ether in these cases. When the importance of this subject is fully realized by the surgeon and general practitioner, and every child before being given an anesthetic is carefully examined for evidences of the status lymphaticus, then death from anesthesia in childhood will be extremely rare.

*Enlargement of the Thymus.*—If attention is directed to the possible presence of this condition by an unexplained chronic dyspnea or general enlargement of lymphoid tissues, enlargement of the thymus, which is the most characteristic sign of this disease, may be demonstrated by physical examination. Blumenreich says that the dullness on percussion produced by the normal thymus gland is in the shape of a triangle, whose base is a line drawn between the sternoclavicular joints, and whose apex is the midsternal line on a level with the second rib. This triangle, however, inclines a little to the left of the sternum at its upper margin. When the thymus gland is enlarged, this triangle of dullness is extended in all directions, but especially to the left of the sternum, and below the clavicle. Careful percussion will, in the great majority of cases, demonstrate enlargement of the thymus. Boggs says the lower border of thymic dullness moves upward when the head is thrown back. The enlarged gland may sometimes be felt in the suprasternal fossa.

Radiography is one of the methods of demonstrating an enlarged thymus. The radiograph shows the shadow of the thymus as continuous with the heart shadow extending up on both sides of the sternum into the neck.

The *spleen, lymph nodes, tonsils, pharyngeal adenoids* and *follicles* at the base of the tongue are commonly enlarged. *Tumor masses* in the neck or in the abdomen may be produced by the agglutination of large lymph nodes. *Itching of the skin* is a common symptom.

A *blood examination* reveals a well-marked chloranemia, the hemoglobin being markedly reduced, the red corpuscles normal in number, but showing many normoblasts and poikilocytes. A marked leukocytosis is commonly present, and the differential count shows a great relative increase in the number of lymphocytes. In one of my cases the blood examination was as follows: Hemoglobin, 65 to 70 per cent.; red corpuscles, fresh preparation, showed considerable poikilocytosis, a few "ameboid" micropoikilocytes, and, on the whole, the red cells were smaller than normal; the stained preparation showed 6 normoblasts to 500 whites counted; white corpuscles, 24,600; red corpuscles, 5,881,250; color index, 0.58. Differential count: polymorphonuclear neutrophiles, 25.6 per cent.; small lymphocytes, 61.8 per cent.; large lymphocytes, 2.6 per cent.; large mononuclear leukocytes, 8.4 per cent.; eosinophiles, 0.6 per cent.; mast cells, 1 per cent.

The child is usually fat, flabby, and to the naked eye presents a well-marked anemia bordering on pallor. The heart is usually rapid and irritable, and may be acutely dilated. These children are non-resistant, malnourished, neurotic, predisposed to convulsive disorders, and frequently succumb to the acute infections.

In rare instances the thymus gland alone, of all the lymphoid tissues of the body, suffers hyperplasia. In such cases the symptom-complex of the status lymphaticus as above given is not complete, the patient suffering only from those symptoms produced by an enlarged thymus.

**Prognosis.**—This condition is fraught with many dangers. Many of these patients die from intercurrent diseases such as the acute infections. Many of the deaths from anesthesia and many of the sudden deaths, especially in childhood, from slight or unascertainable causes are due to this condition. The brilliant results, however, which have recently been obtained by surgery and by the use of the X-rays promise to greatly diminish the mortality from this disease. It should also be remembered that as time goes on the natural physiological atrophy of the thymus gland tends to a spontaneous cure, so that in the milder cases the disease is gradually outgrown.

**Treatment.**—It is my belief that in infants and young children RÖNTGEN RAYS act specifically in the control of the dyspnea and certain other important symptoms of status lymphaticus, and that when this method of treatment is judiciously used and assisted by other therapeutic measures, the symptoms of this disease may be controlled until time, which brings about gradual diminution in the size and function of the thymus, completes the cure.

When the enlarged thymus gland in a case of status lymphaticus is exposed to the influence of the X-rays, we have, as a result of this treatment[1]:

"1. Decrease in size of the hyperplastic thymus, with the disappearance of the cough, stridor, and asthma.

"2. Decrease in size of the enlarged spleen and lymph nodes.

"3. Stimulation of the physical and intellectual growth of the patient.

"4. Rapid disappearance of the marked lymphocytosis which characterizes this disease.

"5. Control of the excessive physiological action of the thymus gland.

"The slight return of the symptoms, stridor, cough, etc., at intervals of three or four months, in one of my cases, and the quick control of these symptoms by one or two exposures to the X-rays indicate that the gradual regeneration of the thymus following the X-ray treatment may be accompanied by a gradual reproduction of the same pathological conditions, hypersecretion, etc., which were present before the treatment was begun. Since the above remarkable results are brought about by the action of the X-rays on the thymus gland, it would appear that the excessive physiological activity of the thymus gland bears the same relationship to status

[1] The author in *The American Journal of Medical Sciences*, October, 1910.

lymphaticus that excessive activity of the thyroid gland bears to exophthalmic goiter. One seems justified in inferring from the above facts that the exciting cause of true status lymphaticus acts primarily on the thymus gland, commonly producing marked hyperplasia of this organ with an increase in or perversion of its internal secretion, and that this increased or perverted secretion is responsible for the general hyperplasia of lymphoid tissues, the lymphocytosis, and general feebleness of constitution which occur in this disease. This inference seems justified by the facts above noted, that the general hyperplasia of lymphoid structures, as well as all of the other symptoms of status lymphaticus, disappear when the X-rays reduce the thymus to normal size and, perhaps of more importance, to normal functional activity."

Alfred Friedlander [1] demonstrated experimentally that any degree of fibrosis of the thymus gland could be produced by the action of the X-ray on this gland and that a thymus thus partially involuted is capable of regeneration.

In 1907 Alfred Friedlander reported a case of status lymphaticus with marked enlargement of the thymus and persistent stridor. Heinecke (quoted by Friedlander) demonstrated the selective action of Röntgen rays on lymphoid tissues, including the thymus, and showed that under their action marked changes occurred in these tissues with a reduction in their size. In a case of status lymphaticus Hochsinger, by repeated exposures, decreased the area of thymic dullness and greatly improved the stridor. In the last few years a number of successful cases have been reported.

The technique of this treatment in my cases was as follows: The tube for the passage of the X-rays had an aperture two inches in diameter, was enclosed in a ray-proof shield, and every portion of the body of the child, except the region of the thymus, was protected from the rays. Injury to the skin was guarded against by filtering the rays through a piece of sole leather. The distance of the tube from the skin was ten inches and the amount of current used 1 milliampère. The character of the tube was high vacuum and well seasoned penetration (Walter 6). The exposure was directly over the thymus gland both anteriorly and posteriorly; the time of exposure in beginning the treatments was three minutes; at the close of the treatments in the fifth or sixth week it was eight minutes; from fourteen to eighteen treatments were given. During the first week from four to six treatments were given of three minutes each both anteriorly and posteriorly; during the second week three treatments of four minutes each anteriorly and posteriorly; during the third week two or three treatments of six minutes each anteriorly and posteriorly; during the fourth week no treatments were given; during the fifth and sixth weeks two treatments of eight minutes each.

Under this treatment the enlargement of the thymus, spleen, and other lymphoid tissues was gradually reduced, the dyspnea and cough

[1] *Archives of Pediatrics*, October, 1911.

slowly improved; the lymphocytosis disappeared, and, although the chloranemia commonly remained, there was great improvement in the physical condition of the patient.

No definite rules can at present be given to govern the administration of the Röntgen rays in the treatment of an individual case. One may say, however, that these treatments should be administered as above outlined over a period of from four to six weeks, provided the patient continues to improve. It is not wise, however, to continue the X-ray treatment after the lymphocytosis has disappeared, even though there may remain a slight amount of dyspnea and some cough, since these symptoms, as a rule, entirely disappear within a few weeks after the discontinuance of the treatment. The prolonged use of the X-rays in these cases may aggravate the chloranemia and otherwise interfere with nutritional processes. In the first course of X-ray treatments it is better to make the mistake of stopping the treatment too early than of continuing it too long. If it be found later, after an interval of some months, that the symptoms of status lymphaticus are returning, two or three treatments with the rays at intervals of a few days will again bring these symptoms under control. In one of my cases it was necessary to give a number of X-ray treatments at intervals of three or four months over a period covering a year and a half from the time the first series of treatments were given.

As an adjunct to the X-ray treatment, FRESH AIR, nutritious and easily digested FOOD, and some preparation of easily assimilated IRON are of the very greatest importance. It is advisable early in the case to give some preparation of organic iron preferably combined with malt extract, and this should be continued long after the X-ray treatment has been discontinued, or until the chloranemia has entirely disappeared. In one of my cases a very marked chloranemia, which remained after the discontinuance of the X-ray treatment, very quickly responded to hypodermic injections of neutral citrate of iron in ¾-grain doses given once a day.

In the management of a case of status lymphaticus it is important to remember that great danger attends the giving of ANESTHETICS, and that sudden shocks to the nervous system, such as result from cold baths, may endanger life, and that it is important to avoid all acute infections, especially those which involve the respiratory tract, since these diseases not uncommonly end fatally in this class of cases.

The amount of EXERCISE which these patients take should be carefully regulated. If marked dyspnea or a dilated heart with rapid and feeble heart action exists, exercise not only aggravates these symptoms, but it may be even dangerous to life.

If syphilis be suspected, ANTISYPHILITIC TREATMENT should be given; if rickets is present, fresh air, cod-liver oil, and a proper diet should be prescribed.

SURGICAL TREATMENT.—The results of thymic surgery have been unusually brilliant; five cases are reported in which this treatment resulted in a cure. All of these cases were aggravated ones, the patient suffering

from extreme dyspnea and other distressing symptoms of status lymphaticus. The operation consists in the complete or partial removal of the thymus gland. As a result of experience it is advised that in infants and young children only the upper portion of the gland be removed, and that the lower portion be drawn up so as to lift it from its position and hold it by stitching to the surrounding tissues. The total removal of the thymus gland in young infants will, it is believed by König and others, interfere with subsequent development. At the present time, however, the indications are that the X-ray treatment will supersede the surgical treatment of this condition.

## CHAPTER LXXI

### DISEASES OF THE SPLEEN: ENLARGEMENT

In infancy the normal spleen varies in length from 4 to 6 or 8 cm. It extends from the mid-axillary line backward, its upper border corresponding with the ninth and its lower with the eleventh rib.

Enlargement of the spleen is very common in infancy, and in many conditions it is of great diagnostic importance. One depends almost exclu-

Fig. 83.—Position in Palpating the Spleen.

sively upon palpation for the diagnosis of enlarged spleen in infancy and childhood. At this age an enlarged spleen can be very readily felt beneath the margin of the ribs in the mid-axillary line. This sign is much more common, much more easily made out, and of much more clinical significance in the child than it is in the adult. Percussion may also be utilized in outlining this organ, but this method is of much greater value in the adult than it is in the child. When the spleen can be readily felt beneath the margin of the eleventh rib, it is either enlarged or displaced downward;

enlargement is very common, displacement very rare. The enlargement may be so great as to almost fill the abdominal cavity; this increase in size occurs downward and toward the umbilicus.

The clinical significance of an enlarged spleen will depend largely upon the cause, and upon the symptom group with which it is associated. In malaria, typhoid fever, status lymphaticus, leukemia, pseudoleukemia of infancy, and tuberculosis (abdominal, intestinal and acute miliary), a well-marked enlargement of the spleen is usually present, and this sign is of great diagnostic value when associated with the other symptoms of these diseases. A moderate enlargement of the spleen, of not so much diagnostic value, is found in syphilis, rickets, gastroenteritis, Hodgkin's disease, sepsis, amyloid disease, chronic malnutritions, anemia, heart disease, peritonitis, Banti's disease and nearly all the acute infections.

Enlargement of the spleen produced by or associated with the above-named conditions is most commonly due to hyperplasia of its lymphoid elements. Passive congestion of the spleen may result from heart disease, cirrhosis of the liver, chronic peritonitis and all conditions that interfere with the portal circulation. Amyloid disease may be caused by suppuration and chronic bone diseases, and is associated with amyloid disease in other parts of the body. Inflammation of the spleen (splenitis and perisplenitis) may be produced by peritonitis, syphilis, tuberculosis and trauma. Displacement of the spleen downward may be produced by pleurisy with effusion, or there may be an actual prolapsus. The latter condition is usually associated with a general enteroptosis of the stomach, liver, kidneys, etc.

Primary splenomegaly is a rare form of splenic tumor due to a hyperplasia of its endothelial cells. It occurs in early childhood and progresses slowly to a fatal termination, lasting for years. The condition was first described by Gaucher and later carefully studied by Vovaird. The splenic tumor gradually increases in size until it may fill the abdomen and produce pressure symptoms of various kinds. There is marked simple anemia, and a pronounced hemorrhagic tendency. There may be bleedings from the nose and gums and subcutaneous hemorrhages may occur.

## CHAPTER LXXII

### DISEASES OF THE THYROID GLAND

### SPORADIC CRETINISM

*(Infantile Myxedema)*

There are two varieties of cretinism, endemic and sporadic. Endemic cretinism, or myxedema, is due to a total or partial destruction of the thyroid gland caused by congenital defects or disease, commonly associated with a goiterous enlargement. It is endemic in certain mountainous

districts in Switzerland, and is characterized by dwarfishness of mind and body and by myxedema of the subcutaneous tissues.

Sporadic cretinism, the common and only form seen in this country, is due to an absence or atrophy of the thyroid gland usually congenital, and very rarely associated with goiterous enlargement. In rare instances myxedema may result from disease of the thyroid gland following acute infections or from the complete removal of this gland by surgical operations. These forms of myxedema, occurring in infancy, may present a clinical picture similar to that of ordinary congenital sporadic cretinism, but these acquired myxedematous conditions are so infrequent in the infant that they may for practical purposes be disregarded. Sporadic cretinism presents a clinical picture in which, as a rule, both mental and physical dwarfishness are more pronounced than in endemic cretinism or myxedema acquired in childhood.

**Etiology.**—The cause of sporadic cretinism is unknown. Heredity, however, is an important factor. Cases may occur in different generations of a family, but rarely do two cases occur in the same immediate family.

**Symptomatology.**—The characteristic symptoms of cretinism usually appear before the end of the first year of life; they may, however, be present as early as the second month, and in less pronounced cases may be delayed to the third or fourth year. Although the disease is usually congenital, the child at birth presents no symptoms; this is perhaps due to the fact that up to the time of birth the child receives its thyroid secretions from the mother, and after birth, although there may be a total absence of the thyroid body, it requires months to develop the characteristic symptoms which result from the absence of thyroid secretions. Because of its insidious onset, it is most important for the physician not only to keep in mind the general syndrome of this disease, but to be ever on the lookout for the early symptoms which announce its approach; an early diagnosis means success in treatment. Mental dullness is usually the first symptom noted; the infant is placid, torpid and presents more or less evidence of stupidity in failing to do the things which a normal child of its age would do. It cannot be interested in toys, or be attracted by things done for its amusement, and not only fails to use its arms and legs in a normal manner, but becomes more clumsy and less apt in this particular than it formerly was. This physical retrogression with an evident lack of intellectuality is accompanied by a vacant expression which characterizes the face, and there slowly develop the characteristic facies and stunted development of the whole body which make the diagnosis plain. The head is large in proportion to the body, the forehead low and narrow, the fontanels are open and may remain so until the child is eight or ten years of age; the face is broad, the cheeks heavy, the nose flat and wide, the eyes are wide apart and the lids may be puffy, the lips are thick and prominent, and the tongue, which is broad and thick, protrudes through the open mouth; this lolling of the tongue which may be accompanied by

drooling is a very characteristic symptom. The child teethes late, and the teeth are imperfectly formed and decay early. A hoarse gutteral cry may be one of the early symptoms.

The hair is coarse, dry and scanty, the eyebrows are almost lacking, the skin of the whole body is pale, dry and cold to the touch. The subcutaneous tissues are heavy, thick, boggy, and to the touch are firm and resistant; small, fatty tumors are commonly present, especially in the lower regions of the neck and the upper part of the back. The neck is short and thick and, on examination, a depression is found where the thyroid body should be located. There is a pronounced anterior curvature of the spine which produces a hollow back and accentuates the protrusion of the large and pendulous abdomen. This anterior curvature of the body is very pronounced when the child is standing, and is exaggerated by the large and apparently protruding buttocks, giving this portion of the body a kangaroo appearance. Umbilical hernia is common, the hips are heavy, and the legs are short and clumsy. These children, as a rule, do not learn to walk until they are four or five years of age, and then have a peculiar waddling gait, handling their bodies and arms in a slow and clumsy manner. The hands are thick, short and spadelike; the hypothenar eminence is especially prominent; the fingers are short, blunt and heavy.

Fig. 84.—Typical Cretin; Age Four Years.

As the child grows older the dwarfed appearance of the whole body becomes more pronounced; a cretin of twenty may not be more than four feet in height, and with this lack of physical development there is a corresponding lack of mental development. It may not be able to speak and may not have sufficient intelligence to avoid uncleanly habits in urination and defecation. A state of absolute idiocy is present in most of these cases. Troublesome constipation is not an unusual symptom; anemia is present, but the blood examination shows nothing characteristic. The temperature is subnormal, registering 95 or 96 in the rectum. The sexual organs are late in their development.

**Differential Diagnosis.**—There are few diseases that present to the eye so repulsive and so characteristic a picture as cretinism; when once seen it is rarely, if ever, forgotten. It can scarcely be confused with any other condition, except, perhaps, Mongolian idiocy. In this condition, however,

the slanting eyes and the Mongolian type of face, the soft smooth skin, the soft, straight, normal hair, the small brachycephalic skull, and the absence of swelling in the tongue and lips should suffice to make the diagnosis plain. If in doubt, however, the therapeutic test will definitely differentiate the two conditions; Mongolian idiocy does not respond to the thyroid therapy.

*Thyroid Insufficiency.*—While the diagnosis of ordinary cretinism presents but few difficulties, it should be remembered that there are a much greater number of cases in which there are degrees of thyroid insufficiency varying from a condition in which the thyroid insufficiency is so slight that it produces few or no characteristic symptoms to the condition, above described, of true cretinism in which the thyroid gland is congenitally absent or atrophied. These cases of thyroid insufficiency present in a modified form and milder degree the symptoms of cretinism. They are undersized and underweight, are lacking to a greater or less degree in mental development, and are grouped in the schools with the "backward" children. As a rule, they have subnormal temperatures.

FIG. 85.—SAME CRETIN; AGE TWELVE, AFTER EIGHT YEARS OF TREATMENT.

She is now fourteen years old and is holding her own in the 5th grade of the Cincinnati public schools. The average age of the pupils of her grade is about four years younger.

**Prognosis.**—The earlier the diagnosis the better the prognosis. If treatment is begun during the first year of life, the child's physical development may be perfect, and its mental development will be almost, but, perhaps, not quite, normal; yet, on the whole, the result in these cases is satisfactory, since they may become useful members of society, attaining a fair degree of intellectual development and acquiring sufficient education to enable them to follow some useful avocation. If the treatment is begun later in the life of the child the results, while striking, are not so satisfactory, and even in cases where the treatment is begun as late as puberty, marked physical and mental improvement may result, but these cases can never be benefited sufficiently to make them self-supporting. Untreated cases remain hopeless, repulsive idiots.

**Treatment.**—In the whole range of medicine there is no more remarkable example of the marvelous curative effect of a therapeutic measure than is furnished by the thyroid treatment of cretinism. In this treatment we have a brilliant example of true specific medication. Previous to the discovery of the specific action produced by feeding the thyroid gland to

cretins, these patients were absolutely beyond the reach of medical treatment; they remained hopeless and helpless imbeciles, fortunately dying of some intercurrent disease before middle life. At the present time, under thyroid treatment, they are slowly transformed into comparatively normal individuals. A cretin that came to me at the age of four years, and who has been constantly under treatment for the last eight years, has, as shown by the accompanying photograph, a fair degree of physical and mental development. At fourteen years of age she is holding her own in the fifth grade of the Cincinnati public schools. The average age of the pupils of her grade is four years younger.

Desiccated thyroids in tablet form are now universally used. They are put up in five-grain tablets, each containing one to two grains of desiccated thyroids. Whatever make of tablet is selected for the treatment of a case, the same should be continued throughout. The dose for an infant under one year of age is ½ grain two or three times a day; it is rarely necessary to give more. For children three or four years of age the initial dose may be 1 grain, and it may be necessary to increase this to 2 grains. It is my belief that much harm may be done by giving too large doses. The most satisfactory results are obtained by the long-continued use of small doses. If the child fails to respond to the above dosage, or if the progress in its physical and mental growth at any time comes to a standstill, then the dose should be gradually and carefully increased. If at any time in increasing the dosage the child shows symptoms of thyroid intoxication, the treatment is to be discontinued for a week or ten days until these symptoms have entirely subsided. Thyroid intoxication is indicated in the young child by pallor, rapid heart action, general nervous irritability, and in older children by headache; fatal syncope may occur. In a case which I saw in consultation over a number of years, very satisfactory progress was made with small doses of desiccated thyroid. The physician, however, yielding to the importunities of the parents, gradually increased the dose until a very severe thyroid intoxication was produced, from which the child never recovered, dying shortly afterwards. In the treatment of these cases, therefore, the physician must be satisfied with the slow and gradual improvement which follows moderate size doses; otherwise he may learn by experience the important lesson that these children can be seriously injured by overdosing with thyroid. I have rarely found it necessary in the treatment of any case to give more than 6 grains of desiccated thyroid in a day, and it has been my experience that the dose which has been found effective in the treatment of the condition in the individual child is the dose that should be continued ever afterward to prevent a return of this condition. In the cases that I have had under observation for six or seven years the treatment has never been interrupted in any case for more than two or three weeks at a time, and this, I think, should be the rule throughout life.

Other treatment may, perhaps, be of little avail, yet it has been my custom in recent years to use iodin and calcium in the treatment of

cretinism. The iodin may be given in the form of syrup of hydriodic acid in ½- to 1-drachm doses, or as iodonucleoids in 1- or 2-grain doses. Calcium may be used in the form of an elixir of the glycerophosphates of lime and soda in from ½- to 1-drachm doses, or in the soluble lactate in 1- to 3-grain doses. The iodin and calcium medication may alternate, or from time to time may be interrupted. I believe that by the use of these drugs better therapeutic results are obtained than by the use of the thyroid extract alone. The change in prescriptions also serves the excellent purpose of keeping up the interest of the mother and patient in the medical treatment, which, if confined to the thyroid alone, after a time becomes monotonous and may lead the mother to conclude that, if no other medical treatment is to be given, she can continue the thyroid treatment herself without medical supervision.

## THYROID INTOXICATION

The thyroid gland is one of the organs which has its greatest functional activity during the early years of life. It furnishes a secretion which exercises such a controlling influence over the body chemistry that, without it, normal growth and development cannot be carried on. This function of the thyroid is so nicely adjusted to the needs of the organism that, as a rule, it furnishes this secretion in quantity and quality accurately adjusted to the purposes it is to serve. In a few instances, however, this gland is congenitally absent; in others its functional capacity is diminished or destroyed by disease or accident; the resulting conditions are known as cretinism and myxedema.

On the other hand, from an increased functional capacity of the thyroid gland we may have an excess of its secretions poured into the body media, producing a well-known group of nervous symptoms. This symptom group may be produced experimentally in man by feeding excessive quantities of thyroid. It is sometimes observed from overdosage in the treatment of cretinism, and it may be observed in exophthalmic goiter, the symptoms of which are at least in part produced by thyroid intoxication. This symptom group is characterized by headache, general nervous irritability, rapid and at times irregular heart action, pallor, cyanosis, great bodily weakness, and a sense of precordial distress. It is my belief that this symptom group in a modified form very commonly occurs in rapidly growing children, and while it may be associated with a slight enlargement of the thyroid, it is in no way related to disease of the thyroid gland or to the development of exophthalmic goiter, later in life. It is simply a thyroid intoxication due to the overaction of this gland. We know that thyroid secretions increase the excitability and stimulate the growth and functional development of the nervous system. It seems very probable, therefore, that since childhood is the period of life when great thyroid activity is an important factor in producing the rapid growth and functional development of the nervous system, it may, when slightly over-

active, so stimulate the nervous system as to produce functional nervous disorders. It may, and undoubtedly does, happen that the amount of thyroid secretion varies with the individual child, and when the secretion is excessive it may produce too rapid growth and development of the nervous system, accompanied by nervous irritability, mental precocity, tachycardia, headache, and other nervous symptoms so commonly observed in the rapidly growing child. If to this symptom group is added a slightly enlarged thyroid, the diagnosis of thyroid intoxication may be made.

**Treatment.**—The recognition of this condition is important. It is transitory, and, after a time, nature adjusts the amount of thyroid secretion more nicely to the wants of the body, and with this adjustment the rapid growth and nervous symptoms disappear. During the period, however, of the thyroid intoxication these children should be protected from overwork, both mental and physical, and in their home life and amusements they should be placed under conditions that will not exaggerate nervous irritability. It may be necessary for a time to remove them from school and to place them under quiet surroundings, which will in no way excite their nervous systems. The bromids may be of value, but the coal-tar preparations are contraindicated in the treatment of this condition.

# SECTION X

## *DISEASES OF THE UROGENITAL SYSTEM*

## CHAPTER LXXIII

### THE URINE

The medical profession has long been fully impressed with the necessity for routine urine examinations in older children, so that this secretion has been studied quite as carefully in the older child as in the adult. Frequent and careful urine examinations in scarlet fever, diphtheria, influenza, and other infections, and especially in convulsive and other nervous disorders of childhood, have long been recognized as of the greatest importance.

The difficulty, however, of obtaining specimens of urine from infants has been a great barrier to its systematic study both in the well and in the sick baby. In recent years medical men have come to realize, more and more, the necessity for routine urine examinations in the infant, and many careful investigations have added greatly to our knowledge. These investigations have shown that serious kidney lesions are less common in the infant than they are in the child, but they have also demonstrated that many heretofore obscure diseases of the genitourinary organs of the infant can be satisfactorily diagnosed only by an examination of the urine.

The following methods have been recommended for collecting the urine of infants: a wide flat sponge, or absorbent cotton, may be held over the external urinary organs by the diaper, and the urine squeezed out of these following urination; a wide-mouthed bottle, or rubber pouch, may be fastened over the external urinary organs with adhesive plaster, this latter method being more satisfactory with the male than with the female infant. Chapin's infant urinal, which is one of the best of a number of devices designed for this purpose, may be fastened over the external genitalia of both male and female infants. When the emergency demands it, catheterization may be resorted to with a small soft rubber catheter—No. 6, American scale; but if the physician has the coöperation of an intelligent nurse, the urine in the great majority of instances may be caught in a suitable vessel as it is expelled from the bladder.

**Quantity of Urine.**—The infant and young child take from four to six times more fluid per kilogram of weight than does the adult, and pass

a correspondingly larger quantity of urine. There may be very great variation from hour to hour and from day to day in the quantity of urine; the amount depending not alone upon the number of ounces of fluid taken, but also upon unexplainable and uncontrollable nervous influences, which may markedly inhibit the quantity one day, and greatly increase it the following day. These fluctuations, especially in young infants, may have no special pathological significance. One may say that from a pathological standpoint the younger the child the less important are these variations. In older children they may indicate, as they do in the adult, an unstable and irritable condition of the nervous system, which in the infant is the normal physiological condition, but in the older child and adult is pathological and commonly due to hereditary or nutritional defects of the nervous system. It is important to remember that suppression of urine (anuria) may occur in infants and young children, and may last over a period of twelve or even twenty-four hours, without indicating a serious pathological condition; in many such instances, the urinary secretion is reestablished without one being able to ascertain the cause of the suppression, but in older children such marked functional disturbances are commonly due to organic disease, although they may be due to such profound functional nervous disorders as hysteria. The following table from Reusing shows the remarkable physiological variations which may occur in the urinary secretion of the healthy infant:

| | Minimum | Maximum |
|---|---|---|
| In the first 24 hours after delivery | 2 c. c. | 61 c. c. |
| 2nd day | 11 c. c. | 145 c. c. |
| 3rd day | 13.3 c. c. | 171 c. c. |
| 4th day | 17.5 c. c. | 179 c. c. |
| 5th day | 22.5 c. c. | 222 c. c. |
| 6th day | 70 c. c. | 280 c. c. |
| 7th day | 93 c. c. | 338 c. c. |
| 8th day | 100 c. c. | 331 c. c. |

The variability in the daily quantity of urine in the infant and young child makes it difficult to compile a table which represents with accuracy the average amount of urine passed. Many careful investigations have been published giving widely varying results. The following table from Jennings is "compiled from the studies of Holt, Churchill, Morse, and other observers," while the carefully prepared tables of Holt, referred to, include the observations of Camerer and other German and French writers:

| Age | Amount in 24 hours | Specific gravity | Urea |
|---|---|---|---|
| First week | 3 to 90 c. c. | 1.010 to 1.004 | 0.07 to 0.66 grams |
| Third month | 200 c. c. | 1.004 " 1.010 | 1.4 " 2.3 " |
| Sixth month | 250 c. c. | 1.006 " 1.012 | 5.0 " |
| Ninth month | 300 c. c. | 1.006 " 1.012 | 7.0 " |
| First year | 400 c. c. | 1.006 " 1.012 | 11.0 " |
| Second year | 450 c. c. | 1.006 " 1.012 | 12.0 " |
| Third year | 500 c. c. | 1.006 " 1.012 | 13.0 " |

| Age | Amount in 24 hours | Specific gravity | | Urea. |
|---|---|---|---|---|
| Fourth year ..... | 550 c. c. | 1.008 to 1.016 | 13.5 | grams |
| Fifth year ..... | 600 c. c. | 1.008 " 1.016 | 14.0 | " |
| Sixth year ..... | 650 c. c. | 1.008 " 1.016 | 15.0 | " |
| Seventh year ..... | 700 c. c. | 1.008 " 1.016 | 16.0 | " |
| Eighth year ..... | 800 c. c. | 1.008 " 1.016 | 18.0 | " |
| Ninth year ..... | 900 c. c. | 1.010 " 1.020 | 19.0 | " |
| Tenth year ..... | 1000 c. c. | 1.012 " 1.020 | 20.0 | " |

**Frequency of Urination.**—Physiological incontinence is the normal condition in infancy. At this time of life urination is purely a reflex act. In the new-born it may not occur on the first or even on the second day, but beginning with the second day the infant usually passes urine two or three times in twenty-four hours, and thereafter, day by day, this increases in frequency until within a few weeks it may be passing urine at hour or even half-hour intervals. After the third month these intervals are gradually prolonged, so that by the end of the second year the bladder of the normal infant may retain urine for from two to four hours, and during sleep even longer. A fair degree of physiological control of this function is obtained about the third year. By this time the child should be able to go through the night without passing urine, and should be able to control this function from four to six hours during the day. Either nocturnal or diurnal incontinence of urine after the third year is to be looked upon as a pathological condition.

## LITHURIA

This condition refers to an excess of the uric acid bodies in the urine, and implies an excess of these same bodies in the blood and tissues. As noted in the above table, the urine of early infancy has a relatively low specific gravity, and contains a comparatively small quantity of urea. On the other hand, uric acid is comparatively very abundant, especially during the early days of life. It is probable that in the fetus, as in cold-blooded animals, uric acid may be one of the end-products of protein metabolism, but after birth this tendency to uric-acid formation rapidly diminishes. This may account for the uric-acid infarcts as well as the temporary anuria followed by the passage of a urine rich in urates, which may occur in the newly-born infant and may even reach a degree of pathological importance. These uric-acid infarcts, as Jacobi has taught, may be a source of great irritation, not only to the kidneys, but to the other urinary organs as well. The irritation they produce in the kidney may be manifested by the presence of albumin and casts in the urine, and by slight hemorrhages which give a tinge of redness to the first urine passed by the infant. An excess of uric acid in the infant may cause a reddish staining of the diapers, and in older children a brick dust precipitation may occur when it is allowed to stand. The ureters, bladder, and urethra may be irritated by the uric acid in passing, and, as a result, the urine may contain large numbers of epithelial cells, leukocytes, red blood corpuscles, and

mucus. Infrequently small renal calculi may form, and, in their passage through the ureters, may cause renal colic, or they may produce great distress by becoming impacted in the urethra. There may be a tendency throughout childhood to recurring attacks of acid urine in which the urates are in great excess; these attacks, as a rule, occur in thin, nervous, irritable, quick-witted children. During the attack the general nervous irritability of the child is greatly exaggerated, the urine is retained as long as possible, and then passed with a fit of crying. The urine in some of these cases is so irritating that the external genitalia become swollen and red, and in female children a mild vulvovaginitis may result. These attacks last for a number of days and then pass off, the urine again returning to normal.

## INDICANURIA

Urinary indican is almost, if not quite, entirely derived from the bacterial fermentation of the proteins of the food. Some observers believe that a small percentage of it may result from the disintegration of albumin in the tissues. However this may be, medical observers are for the most part agreed that this latter source is so unimportant that it may be considered negligible from a pathological standpoint. Indican in the urine is therefore a sign of bacterial disintegration of proteins in the intestinal canal, and the extent of this form of fermentation may, in most instances, be largely determined by the quantity of indican in the urine.

Herter has called attention to the fact that indolaceturia (indolacetic acid in the urine) is also produced by the bacterial fermentation of proteins in the intestinal canal, and that there is a "reciprocal relation between the formation of indolacetic acid and indol," that is to say, when the one is present in large quantities the other will be found in smaller quantities. From this it would appear that both urinary indican and indolacetic acid are evidences of bacterial fermentation of proteins in the intestinal canal.

Indican is usually absent in the urine of normal breast-fed infants, but is found, as a rule, in small quantities in the urine of all other individuals. It has pathological significance only when it occurs in excess. The quantity is generally increased in well-marked constipation and in all gastrointestinal diseases. The degree of indicanuria, in many instances, marks the severity of the gastrointestinal intoxication. Pronounced indolaceturia may also be an indication of intestinal toxemia. In the routine examination of urine, therefore, the approximate quantity of indolacetic acid as well as indican should be determined; an excess of these bodies in the urine is very commonly associated with albumin, occasional hyalin casts, and an excess of oxalate of lime crystals.

## HEMATURIA

Attention is usually called to this condition by the color of the urine, which may be flesh-colored, smoky, brownish, or reddish; when the latter, it may vary from the faintest tinges to a blood-red color, depending on

the quantity of blood in the urine. It should be remembered that in alkaline urine a comparatively small quantity of blood may produce a bright red color. It can be differentiated from hemoglobinuria by a microscopical examination, which reveals the presence of red blood corpuscles in large numbers. Hematuria may occur under a great variety of conditions and be produced by disease or injury of any part of the genitourinary system. In newly born infants the urine passed during the first days of life may be tinged with blood, as a result of irritation from uric-acid crystals and infarcts; this form is transitory and has slight pathological significance. Calcium oxalate crystals, when present in excess, may also produce hematuria; this may sometimes result from the eating of rhubarb, tomatoes, and asparagus; this form is rare and is largely a matter of idiosyncrasy; it is also transitory and has little pathological significance. Infantile scurvy is a cause of hematuria, and in severe cases of this disease red blood corpuscles may almost always be found in the urine, and well-marked hemorrhages may occur. Hematuria may be present as a symptom of sepsis, hemophilia, purpura hemorrhagica, leukemia, severe forms of variola, malaria, scarlet fever, acute nephritis, calculus, tuberculosis, malignant disease, cystitis, neoplasms, and acute inflammation of the urethra (gonorrhea). It may also be produced by certain drugs, such as turpentine, cantharides, and large doses of chlorate of potash.

The source of the blood in the urine cannot always be readily determined. An examination of the external genitalia of the female will discover whether the urine was contaminated after leaving the urethra. If the hemorrhage comes from the kidney, epithelial and blood casts, together with renal epithelium, can, as a rule, be found, and in these cases the quantity of albumin is out of proportion to the quantity of blood. If the blood comes from the bladder it commonly follows the urine and is passed with pain and tenesmus; if the blood precedes the urine its source is the urethral canal. In all cases of hematuria the concomitant symptoms are important in determining the source of the blood.

**Treatment.**—This will depend upon the cause. Hematuria is a symptom of some constitutional disorder or local disease of the genitourinary tract, and the treatment of the causative condition is therefore the treatment of the hematuria. The symptom itself rarely demands special treatment. Fluid extract of ergot or ergotin may be given where the hemorrhage is excessive, and absolute rest in bed should be insisted upon. If the bleeding point can be reached by the injection of adrenalin solution into the bladder the hemorrhage may be controlled in this way.

## HEMOGLOBINURIA

Hemoglobinuria is due to an extensive disintegration of red blood cells, which is followed by the appearance of blood pigment in the urine; this pigment, however, is commonly methemoglobin, rather than unchanged hemoglobin.

The diagnosis is made by the urine findings. The color of the urine varies from a pale red to a very dark red, depending upon the quantity of hemoglobin it contains. The amount of albumin present also depends upon the quantity of hemoglobin. Microscopic examination reveals the blood pigment in amorphous granules, masses, or casts, and hemoglobin crystals may be found, but the important diagnostic feature is the almost complete absence of red blood cells. Hyalin and granular casts are present, and renal epithelium and calcium oxalate crystals are usually found. As the patient convalesces from the attack, the urine gradually loses its red color and the above microscopic findings disappear.

**Etiology.**—Hemoglobinuria is usually an expression of some severe toxemia. It may occur in septic conditions, scarlet fever, malaria, erysipelas, and severe forms of jaundice. It may be produced by certain poisons, such as phenol, naphthol, arseniuretted hydrogen, toluene-diamin, and large doses of chlorate of potash. It may also result from the transfusion of blood, or the injection of a foreign serum.

There are, however, two rather clearly defined types of hemoglobinuria which demand special consideration, namely: Epidemic Hemoglobinuria of the New-born, and Paroxysmal Hemoglobinuria.

### WINCKEL'S DISEASE

Winckel's disease, or epidemic hemoglobinuria of the new-born, is a rare form of hemoglobinuria, probably due to sepsis. It makes its appearance during the first few days of life in apparently healthy infants. It has been observed in epidemic form, particularly in institutions, where the bath water is regarded as a method of infection. An early symptom is cyanosis, which, together with jaundice and hemoglobinuria, forms a striking clinical picture. The disease is marked by profound constitutional symptoms, great depression, a feeble, rapid pulse, cold extremities, and is sometimes associated with diarrhea and vomiting. Jaundice develops early, and is usually intense within twenty-four hours. The urine findings are the same as above described. The disease runs a rapid and fatal course, commonly terminating within two or three days. Convulsions and coma may be the terminal symptoms. The diagnosis of this form of hemoglobinuria is made by the urine findings, the age of the infant, and the profound constitutional symptoms above noted.

### PAROXYSMAL HEMOGLOBINURIA

Paroxysmal hemoglobinuria is a chronic intermittent form which may come on in childhood and recur in paroxysms throughout the life of the individual. In other instances it disappears without apparent cause as the patient grows older.

**Etiology.**—Syphilis and malaria are believed to be important predisposing factors in many cases. Exposure to cold is the most common

exciting cause of the individual attacks. They are therefore much more usual during the winter months. Contact with cold water is a very common exciting cause. The individual attacks may begin with a chill, which may be followed by a rise of temperature. Cyanosis, general depression, nausea, and abdominal pain may be present, and the skin may be slightly jaundiced. The urine findings are the same as those above noted. The attack is of short duration; it passes off within a few hours and the urine rapidly becomes normal. The frequency of these attacks varies greatly; in the winter they may recur at short intervals; during the summer they may be separated by months.

**Prognosis.**—The prognosis, as far as life is concerned, is good. This disease may not shorten life, and some cases recover after reaching adult life.

**Treatment.**—When due to sepsis, or the various acute infectious diseases, these causative conditions are to be treated rather than the hemoglobinuria. In the periodic form antisyphilitic and antimalarial treatment should be tried, and if benefit follows they should be continued. Patients suffering from this disease should also live in a mild and equable climate, so as to avoid sudden chilling of the surface of the body. Cold baths and other exciting causes, which in the individual case are known to precipitate the attack, should be avoided. Martin H. Fischer believes that we can relieve the signs and symptoms of paroxysmal hemoglobinuria by increasing the salts in the diet. Sodium chlorid should be given in some form in every case of hemoglobinuria.

## ACETONURIA

Oxybutyric acid is the antecedent body from which diacetic acid and acetone are formed. These three substances constitute the acetone group, and their appearance in the urine is indicative of a definite form of acid intoxication, which is associated with a large number of diseased conditions. Oxybutyric acid is formed by the disintegration of fat and protein molecules under unfavorable conditions of oxidation. Von Noorden believes that carbohydrate molecules furnish some oxygen to the fat and protein molecules, as they are disintegrated in the normal processes of body metabolism, and that the absence of carbohydrate molecules results in the imperfect oxidation of the disintegrating fat and protein molecules, and thereby causes the formation of oxybutyric acid. According to this view, the presence of oxybutyric acid in the blood is always due either to a deficient intake of carbohydrate foods or to some perversion in the carbohydrate metabolism, which makes it impossible for them to exercise their oxidizing influence on the disintegrating fat and protein molecules. It is my belief that this form of acid intoxication is due either to a deficient absorption of carbohydrate food or to functional or organic diseases of the liver, which interfere with its glycogenic function. While oxybutyric acid is the most important of the acetone series, it cannot be demonstrated

in the urine by simple clinical tests, so that for clinical purposes the urine is examined for either diacetic acid or acetone, and the presence of either of these bodies is an indication that this form of acid intoxication exists. Oxybutyric acid is the antecedent body; this is oxidized into diacetic acid and then into acetone, so that for clinical purposes the demonstration of one of the series is all that is necessary. *The oxidizing processes here referred to are so rapid that acetone is the first of these bodies to appear in the urine, and the subsequent appearance of diacetic acid, and later of oxybutyric acid, indicates failing powers of oxidation and therefore a more severe form of autointoxication.*

This form of acid intoxication may produce deleterious results: First, by the direct toxic action of the acids formed; second, by removing alkaline bases, such as calcium, potassium, sodium, and magnesium, which are necessary to the normal processes of metabolism; third, by bringing poisonous alkaline bases, such as ammonium, in large quantities into the blood (Rachford); fourth, by separating $CO_2$ from its bases, and thus producing carbonic acid poisoning (Herter).

The acetone group may appear in the urine in many pathological conditions, and it is found much more frequently in children than in adults. These bodies are commonly observed in diabetes, malignant disease, prolonged fevers, starvation, gastrointestinal disorders, recurrent vomiting, other forms of severe and prolonged vomiting, nervous disorders, migraine, bronchopneumonia, influenza, severe malnutrition, and in poisoning from atropin, lead, morphin, antipyrin, and chloroform.

**Diagnosis.**—The diagnosis of this form of acid intoxication is made by the finding of one or more of the acetone bodies in the urine. In this examination it is important to remember that the reaction with a solution of ferrous chlorid, which is commonly used to demonstrate the presence of diacetic acid, may be unreliable if the patient has been taking a salicylate, since this drug, when excreted by the urine, gives a reaction similar to that of diacetic acid. If the acetone group is found in large quantities in the urine it is an indication of profound metabolic disturbances, one of the results of which is the presence of these bodies in large quantities in the blood. This form of acid intoxication, however, is not characterized by any well-defined clinical picture. By some writers it is believed that *dyspnea, increased pulse rate, somnolence, and coma,* which occur in many of these cases, are either directly or indirectly due to the acetone bodies. In all of these cases there is an increase in the urinary ammonia, and in some instances a decrease in the alkalescence of the blood.

**Prognosis.**—The prognosis depends upon the cause. If acetonuria appears as a symptom of advanced diabetes or cancer, the prognosis is unfavorable, but even in these cases one may temporarily cause to disappear the coma and other symptoms associated with the acidosis. In the acetonuria, which occurs in acute malnutrition, intestinal disorders, fevers, and "recurrent" and other forms of severe vomiting, the prognosis is nearly always good. There is a form of acidosis occurring in children

under three years of age associated with intestinal disturbance and dyspnea which may terminate fatally.

**Treatment.**—For the immediate relief of an attack of acidosis, from 2 to 5 grains of calomel, combined with bicarbonate of soda, should be given, and should be followed by a saline laxative. Immediately afterward the administration of alkalies in large doses should be begun. The alkaline treatment, which consists in the giving of bicarbonate, or benzoate of soda, should be continued for some time, in smaller doses, after the acetone bodies have disappeared from the urine. During convalescence, and for the prevention of these attacks, fresh air, preferably out-of-door air, is indicated, and this should be continued throughout the twenty-four hours, the patient either sleeping out of doors or with wide open windows. Carbohydrate foods are especially indicated, and should always be given when the condition of the patient will permit. Exercise out of doors is to be recommended in all cases where the individual is strong enough to be benefited by it. Where the physical condition of the patient is such that outdoor exercise cannot be indulged in, general massage is to be recommended. A more detailed account of the treatment of acidosis is given in the chapter on Recurrent Vomiting.

## ALBUMINURIA

The presence of albumin in the urine is not always an indication of structural disease of the kidney. On the other hand, it is now a well-recognized fact that transient forms of albuminuria may occur, even associated with an occasional hyalin or epithelial cast in conditions other than true nephritis. It is necessary, therefore, that these forms of albuminuria should be carefully studied in order that they may not be confused with nephritis, and it is even more important that cases of true nephritis should not be mistaken for these more innocent forms of albuminuria. The following forms of albuminuria may be recognized: Physiological, orthostatic, toxic, and nephritic.

### PHYSIOLOGICAL ALBUMINURIA

This is the transient, or intermittent, appearance of albumin in such quantities that it may be easily discovered by ordinary tests in the otherwise normal urine of a healthy child. It is comparatively unimportant and deserves but passing notice. It may result from the following causes: excessive muscular exercise, such as bicycling, foot-racing, and jumping the rope; emotional excitement, such as may result from masturbation, or pseudomasturbation—this form has little or no pathological significance; sudden and prolonged chilling of the surface of the body, such as may come from a cold bath, the albumin from this cause readily disappears as the child reacts, and may be due either to nervous shock or temporary congestion of the kidney; overfeeding with albuminous food, this cause is sometimes associated with injuries to the metabolism from

the inability of the excretory organs to eliminate the excess of foreign albumin in the circulating media of the body. In each of the above instances the question may arise as to whether the term physiological can properly be used to designate these albuminurias, but certain it is that they may occur entirely apart from disease of the kidneys, and that their pathological importance is so slight that they can scarcely be classified under any other heading. The transient appearance of a faint trace of albumin or an occasional hyalin cast may have no pathological significance; in fact these findings are very common in the urine of normal children.

The albuminuria of the new-born has been designated as physiological. Much attention has been given to this form by German writers, all of whom are agreed that it occurs in a large percentage of infants during the first week of life, and in a few may persist until the end of the second or third week without being considered a pathological process. It is most frequently found during the first and second days of life, but the percentage of infants having albuminuria rapidly diminishes after the third day, so that if it persists to the end of the second week it is perhaps wise to consider it a pathological process, unless it can be shown to belong to one of the other forms of physiological albuminuria above mentioned. In some instances the albuminuria of the new-born may be due to incomplete functional development of the kidney, which results in a defect in its filtering function; or in others it may result from the irritation of uric acid crystals and infarcts, which irritate the tubules of the kidney to such an extent that not only albumin appears but occasional hyalin and epithelial casts are present; or, again, it may be toxic, due to the irritation of the very vulnerable kidney of the newly born infant by intestinal and other toxins. Whatever may be the cause of these forms of albuminuria in the new-born, they are of no pathological importance, since the urine quickly clears and leaves the kidney in a normal condition. For this reason they can scarcely be classified as pathological.

### ORTHOSTATIC ALBUMINURIA

This form was first described by Pavy, under the name of Cyclic Albuminuria. Since his observations, the condition has been very extensively studied by many observers, and Heubner proposed for it the name Orthotic, or Orthostatic, Albuminuria, which term expresses the important fact in its etiology, that the albuminuria is postural, coming on when the patient is on his feet and disappearing when he assumes the recumbent posture. Pavy used the term cyclic to express practically the same condition; he found no albumin in the early-morning urine, while that passed during the day always contained albumin. This cycle which Pavy described, was later found to be due not to the particular time of day when the urine was secreted, but to the upright position of the patient during the day and the prone position during the night. No albuminous cycle occurs in these cases as long as the patient is confined to bed; under this condition both the night and day urines are free from albumin.

**Etiology.**—This is a pathological, not a functional or physiological, form of albuminuria. It is not, however, a symptom of nephritis, and, in the great majority of instances, does not lead to actual disease of the kidney. The fact should be kept in mind that all forms of pathological albuminurias, including the toxic and nephritic varieties, are aggravated by the upright position, and these forms must therefore be carefully differentiated from true orthostatic albuminuria.

Heredity plays an important rôle in the etiology of this condition. A family predisposition is not infrequently found. The exact character of this heredity is unknown, but a gouty and neurotic inheritance is very commonly present. The hereditary defect, however, is perhaps a certain degree of functional disability on the part of the renal epithelium, which permits the albumin of the blood to filter through under slightly abnormal conditions. The fact that the upright position is the all-important factor in producing this form has led to the belief that some circulatory disturbance of the kidney, which is aggravated by this position, is the determining factor. Some observers have been able to demonstrate displacement of the kidney in a considerable percentage of these cases; in the great majority of instances, however, the kidney is in normal position. As this disease appears most frequently between the ages of six and fourteen, during the rapid growth of the child, it has been suggested that the instability of the abdominal viscera during this period is one of the causes of the disturbed circulation in the kidney. Jehle believes that lordosis of the lumbar vertebræ is an important etiological factor in many of these cases. This disease, or rather the pathological condition with which postural albuminuria is associated, is aggravated by anything that lowers the vitality of the child. Neurotic disturbances, functional incompetency of the liver, acute illness, and, in short, all the conditions which are responsible for physiological and toxic albuminuria will aggravate orthostatic albuminuria.

**Symptomatology.**—While, in a few instancees, there is nothing to mark this condition except the urine findings, in the great majority of cases there are more or less well-marked constitutional symptoms. My experience is in accord with that of Longstein, Heubner, Still, and others, that patients suffering from orthostatic albuminuria are physically below par; they are commonly nervous, anemic, have dark circles under their eyes, are easily exhausted, and suffer frequently from headaches and abdominal pain. I have observed that many of them have feeble digestive capacity, and suffer from fermentative disturbances in the intestinal canal; indican and oxalic acid are frequently found in excess in the urine, and not infrequently the albuminuria is increased or diminished in proportion to the amount of these ingredients.

**Urine Findings.**—The diagnosis can be made only by repeated careful examinations of urine, passed under various conditions of diet and posture. The urine secreted while the patient is lying down is normal, containing neither albumin nor casts, but the urine passed after the patient has as-

sumed the upright position contains albumin, sometimes in large quantities. In a patient whom I have had under observation since 1903 and who has now entirely recovered, albumin was often present in the early months of the treatment in quantities of ½ per cent., and could at all times, when she was in an upright position, be easily demonstrated by the ordinary clinical tests. Besides the albumin in these cases, one not infrequently finds an occasional hyalin cast, and in rare instances an occasional epithelial or leukocyte cast; cylindroids are also occasionally present; leukocytes, squamous epithelium, and crystals of calcium oxalate, and uric acid are not uncommon. I have carefully studied the influence of exercise on these patients and have found that it does not increase the albuminuria. They will have more albumin in their urine when they are confined to the house, sitting and walking about, engaged in ordinary indoor occupations, than they will if made to take exercise in the open air. Walking, horseback riding, golf, and other outdoor exercises diminish rather than increase the albuminuria. A milk diet will not diminish the albuminuria; on the other hand, it will, if prolonged, aggravate it. These are important points in the differential diagnosis of this condition from nephritis.

**Prognosis.**—The present status of our knowledge of orthostatic albuminuria makes it advisable that the physician render a guarded prognosis. There is no question but that some of the cases, which have been carefully studied by competent observers, have ultimately terminated in nephritis. On the other hand, there can be no question but that many of these cases, after years of albuminuria, permanently recover. On the whole, it may be said that the prognosis is favorable, and that, under proper care, most of these cases get well.

**Treatment.**—These patients should lead an outdoor life in fresh air, and take a moderate amount of physical exercise. In beginning the treatment, it is important that the amount of exercise prescribed should fit the individual case, otherwise the child may be overfatigued, but as health and strength return no limitation should be placed upon the outdoor exercise other than that overfatigue should be avoided. They are never benefited by rest in bed; this is a measure which is only justifiable for the purpose of making the diagnosis. An exclusive milk diet is not only inadvisable, but, if prolonged, does harm. A liberal diet of milk, cereals, vegetables, fruits, and a moderate amount of meat and eggs should be prescribed. I have not been able to satisfy myself that a moderate amount of albuminous foods, such as meat and eggs, exerts any unfavorable influence. The restriction therefore along this line should be that these albuminous foods are not taken in excess. I have further convinced myself that these cases are benefited by the *rigid exclusion of sweets* from the diet. Wines and malt liquors are also contraindicated; rich and highly seasoned foods are not to be recommended. In two instances I have been convinced that rhubarb aggravated the albuminuria. Still advises that this vegetable, together with tomatoes and asparagus, be excluded from

the diet. I further advise the drinking of water between meals, and, above all, that the food of the patient should be selected to suit his individual digestive capacity. Intestinal fermentation is to be carefully avoided, and the patient's whole life is to be so directed that his general health may be improved and his power of resistance increased. The albuminuria cannot be influenced by the medical or other therapeutic measures which favorably influence the albuminuria of Bright's disease.

## CHAPTER LXXIV

### ACUTE NEPHRITIS

From a pathological standpoint the term acute nephritis comprehends a variety of degenerative and inflammatory changes which are produced in the kidney by the action of the toxins and microorganisms. The degenerative changes, which are confined chiefly to the epithelial cells, are for the most part caused by the action of toxins. The inflammatory changes, which include not only degeneration of epithelial structures but the infiltration of the kidneys by newly formed cells, and perhaps later by increased growth of connective tissue, are produced by the combined action of bacteria and their toxins. Martin H. Fischer believes that an abnormal production or accumulation of acid in the kidneys, acting on their colloidal structures, explains the pathological findings and the resulting symptoms of nephritis. In the present state of our knowledge, however, we are not prepared to clearly differentiate either from a clinical or pathological standpoint between a purely degenerative (toxic) and an inflammatory (mycotic) nephritis. It is a recognized fact that, in some instances, both the so-called degenerative and inflammatory lesions may be produced in the kidney by the action of bacterial and other toxins without the kidney itself being infected with bacteria. On the other hand, it is generally believed that in a large percentage of the so-called true inflammatory processes of the kidney, and especially in those of a severe type, the kidney itself is infected with pathogenic microorganisms. While we recognize the existence therefore of a toxic and of a mycotic form of nephritis, we cannot, from a clinical standpoint, clearly differentiate these conditions. We know, however, that in the vast majority of instances the disease begins, and in a large percentage of cases remains a purely degenerative process produced by the irritating action on the kidney of toxins. We know also that in a considerable number of these cases the degenerative changes thus produced predispose this organ to infection, and that a mycotic nephritis is added to the toxic nephritis, and when this occurs the gravity of the disease is greatly increased. We know also that a small percentage of the cases of nephritis may be classed as primarily mycotic, that is to say, the kidney is attacked by pathogenic microorganisms before degenerative changes in the epithelial structures have

prepared the way for this infection. These cases are usually of a very severe type, and occur either as so-called idiopathic or primary cases, or during the first week of one of the acute infections, such as scarlet fever, diphtheria, and influenza. The terms parenchymatous, tubular, and glomerular nephritis have been used to describe various localizations of the ordinary toxic, or degenerative, form, while the terms acute diffused nephritis and acute productive nephritis have been used to describe the more severe inflammatory types. From a clinical standpoint, however, this classification is of no assistance. For these reasons we shall speak of the toxic and mycotic forms of acute nephritis, recognizing that they are frequently commingled, and, even when distinct, may be difficult of differentiation.

## TOXIC NEPHRITIS

(*Toxic Albuminuria*)

The severity of this form depends upon the virulence of the toxin, the length of time it acts on the kidney, and the part of the secreting structure attacked. These cases may, by bacterial infection, be converted into a true mycotic nephritis, but in the great majority of instances they run a benign course, the albumin and casts disappearing as the patient recovers from the disease, systemic or intestinal, which is producing the toxemia.

**Etiology.**—This form of nephritis is commonly produced by scarlet fever, pneumonia, influenza, septicemia, malaria, rheumatism, cerebrospinal meningitis, and gastrointestinal infections. Chapin, Morse, and others have called special attention to the frequency of albuminuria in pneumonia and cerebrospinal meningitis. Chapin reported "a series of 57 cases of pulmonary diseases, such as severe bronchitis, pleurisy, and pneumonia, that gave the following results: 49 had albumin in the urine, thus noted; trace, 13; faint trace, 30; heavy trace, 6; 32 cases had casts present, either hyalin, granular, epithelial, or mucous." In infancy, disease of the gastrointestinal tract, as Kjelberg demonstrated, is the most common cause of this condition; Koplik, Morse, Chapin, and many others have also noted this fact. In Koplik's series of 25 cases of gastroenteritis all but 4 had albuminuria, and in more than one-half casts were found. In Chapin's 86 cases of gastrointestinal disease, albumin was present in 75, and casts, hyalin, granular, epithelial, or mucous, were present in 37. It is a notable fact that in cases of gastrointestinal origin the albumin in the urine rises and falls with the amount of indican. This, however, does not necessarily mean that the indican produces the irritation of the kidney, but it does indicate that the products of the intestinal fermentation of albuminous foodstuffs, of which indican is one, are largely responsible for this irritation.

Severe anemia, scurvy, icterus, and diabetes may be associated with albuminuria, the cause of which is perhaps toxic. Migraine, "recurrent vomiting," and other autointoxications may produce albuminuria; the

writer, many years ago, called attention to the presence of transient albuminurias occurring in connection with these toxic states.

A few drugs and chemicals, such as turpentine, chlorate of potash, carbolic acid, cantharides, arsenic, and phosphorus, may, when taken in excess, produce a toxic nephritis. In some of these cases the irritation to the kidney may be so great as to produce a violent inflammation associated with severe and dangerous constitutional symptoms.

**Symptomatology.**—In the great majority of these cases the diagnosis must be made almost exclusively by the urine findings, since no constitutional symptoms referable to the kidney lesions are present. This is especially true in the toxic albuminurias which occur in infants and young children suffering from catarrhal diseases of the intestinal canal and respiratory tract, as well as in older children suffering from some form of autointoxication, or one of the acute infections. In these cases the constitutional symptoms of the original infection are present, and the kidney lesion is, as a rule, accidentally discovered by an examination of the urine. It must, however, be remembered that even the purely toxic forms of nephritis may occasionally be associated with kidney lesions so severe as to produce uremic and other constitutional symptoms. The symptom group occurring in these cases is similar to that detailed under the heading Mycotic Nephritis.

The urine findings upon which the diagnosis is made are as follows: albumin, usually not a great amount; a small number of hyalin and epithelial casts; leukocytes, epithelial cells and mucus. The quantity of the urine may not be diminished, but the specific gravity is usually high, sometimes above 1,020.

**Diagnosis.**—If the physician keeps in mind the fact that in all the above-named conditions mild forms of toxic (degenerative) nephritis are very common, and that severe forms of acute mycotic nephritis are comparatively rare, he will then not easily be misled by the urine findings above noted. The small amount of albumin, the comparative scarcity of casts, and the absence of all constitutional symptoms referable to the kidney would justify the diagnosis of a simple toxic nephritis.

**Prognosis.**—The great majority of these cases recover without apparently influencing the course of the disease with which they are associated. Rarely, however, the toxins may be so virulent or their action on the kidney may be so long continued as to produce severe and dangerous forms of nephritis. In some instances, also, the toxic nephritis may predispose to, and later be associated with, a true mycotic nephritis.

**Treatment.**—The object of all treatment is to protect the kidneys, whose secreting structures are beginning to break down under the elimination of excessive quantities of toxins. This may be accomplished in three ways, namely: diminishing the toxemia, eliminating the toxins through other organs than the kidney, and protecting the kidney by a non-irritating diet. In cases associated with gastrointestinal intoxication much can be done to diminish the toxemia by laxative medication. So that in every instance,

whatever may be the cause of the toxemia, a cathartic should be given, and the primary disease producing it should then receive appropriate treatment, especially with reference to diminishing and controlling, as quickly as possible, the general toxic condition. A most important part of the treatment consists in eliminating the toxins through other channels than the kidney. This may be done by saline cathartics, by warm baths, and by the administration of large quantities of alkaline water. Perhaps most important of all is the dietetic treatment. Where the gastrointestinal canal is not involved and the question of diet pertains simply to the protection of the kidneys, the food given should be similar to that recommended in acute mycotic nephritis, milk and cereals predominating. In many of these cases, especially those associated with pneumonia and acute septic conditions, the advisability of the administration of alcohol may arise, the causative condition perhaps demanding alcoholic stimulation, which is contraindicated by the presence of albumin and casts in the urine. The urgency of the symptoms of the causative condition may, in some of these cases, demand that the dietetic treatment of the kidney irritation should be temporarily neglected.

## ACUTE MYCOTIC NEPHRITIS

(*Acute Diffuse Nephritis, Acute Productive Nephritis, Acute Bright's Disease, True Nephritis*)

This is an acute non-suppurative inflammation produced by bacteria and their toxins. It may involve any part or all of the kidney structure.

**Etiology.**—It commonly occurs as a complication or a manifestation of the acute infectious diseases; in these conditions the primary inflammation is usually started by the irritation of bacterial toxins, which are eliminated in large quantities through the urine. The kidney structures thus inflamed are in no condition to resist the pathogenic microorganisms, which are also being excreted by them, and, as a result, a mycotic infection is added to the toxic nephritis. This secondary mycotic nephritis usually occurs late in the causative disease, ofttimes when the patient is believed to be convalescent from the acute infection. While secondary or late mycotic nephritis is one of the most severe and dangerous complications of the acute infectious diseases, yet it is nothing like so dangerous as the form of acute nephritis which is produced by a primary mycotic infection of the kidney. Acute nephritis of this type occurs during the early acute symptoms of the causative infection, and becomes at once the most serious and dominating symptom-complex of the disease; it not infrequently assumes the acute hemorrhagic form. The so-called idiopathic or primary cases of acute nephritis, which occur in young children, and whose etiology is so obscure, represent a small percentage of this class of cases. The kidney is the organ primarily attacked, or the preliminary symptoms of the influenza, tonsillitis, "cold," or other causative factors are so slight as to be overlooked until a sharp and severe mycotic nephritis calls at-

tention to the infection. The term, primary nephritis, may therefore, perhaps, be used in describing these cases, but the term "idiopathic" is misleading and should be discarded.

Scarlet fever, of all the acute infections, is most commonly followed by acute nephritis. In this disease the skin is largely put out of function by the acute dermatitis, and the kidney is therefore called upon to do an enormous amount of work, under which it very frequently breaks down, and an acute toxic or mycotic nephritis results. In addition to this, the scarlatinal poison has a selective action on the kidney, and especially on its glomeruli, hence the term glomerular nephritis. Scarlatinal nephritis usually occurs late in the disease, and has a very gradual or insidious onset. It may, however, occur during the first week; this form, which is comparatively rare, is sudden in its onset, violent in its course, and not infrequently assumes the hemorrhagic type. Nephritis may also be produced by influenza, diphtheria, enteritis, variola, rheumatism, malaria, tonsillitis, typhoid fever, pneumonia, septicemia, measles, varicella, and congenital syphilis. It should also be remembered, as previously noted, that certain drugs and chemicals, such as turpentine, chlorate of potash, carbolic acid, cantharides, arsenic, and phosphorus may, in rare instances, produce violent inflammations of the kidneys, and that the toxic nephritis thus set up may terminate in a true mycotic nephritis.

The influence of cold as a cause of nephritis has probably been greatly exaggerated; it may be a contributing factor. It is difficult to conceive how exposure to cold as an independent factor may produce nephritis, but it is easy to understand that it may be very important if the child be suffering from some acute infection, that is to say, if the kidney be already irritated by the presence of toxins or microorganisms.

**Symptomatology.**—Clinical experience has taught me to believe that one may determine, by the character of the onset and by the violence of the early symptoms, whether the disease is primarily toxic or mycotic in its origin. The toxic cases are more insidious and commonly present no constitutional symptoms; if, however, bacterial infection is added to toxic irritation of the kidney, pronounced and often violent constitutional symptoms are gradually added to the symptom group. Cases of this character occur as a late complication of one of the acute infectious diseases. The primary mycotic cases on the other hand are comparatively violent in their onset, and occur with the initial symptoms of the causative disease. Under this heading one may include all those cases of nephritis which occur during the first week of the acute infectious diseases, and probably all the so-called "idiopathic" or primary cases in which the exciting cause is not apparent. These so-called primary cases have been carefully studied and described by Holt and others; they occur in young children, are violent in their onset, and run a severe and usually fatal course.

There are three early manifestations of acute nephritis, any one of which may suggest its onset: First, edema, especially of the face; second, certain nervous uremic manifestations, such as headache, vomiting, drow-

siness, and disturbances of vision; third, the appearance of albumin and casts in the urine. The physician should therefore be ever on the lookout for these signs of nephritis, especially when he is treating one of the acute infectious diseases of childhood.

Dropsy is one of the common symptoms of nephritis, developing early in the disease and usually first showing itself as a slight edema of the face. The eyelids may be puffy, and the whole face may gradually become slightly edematous, presenting a peculiar pallid color; later the same puffy edematous condition of the skin may appear in the hands and over the lumbar region. In some instances the dropsy may spread, producing a general anasarca; the whole body including the legs, arms, back, abdomen, and scrotum, may be swollen and edematous, and the peritoneal and other serous cavities may contain large quantities of fluid. These cases of extensive dropsy occur most commonly in scarlatinal nephritis, but marked dropsy may also be associated with nephritis due to other causes. The extent of the dropsy is of comparatively little value in prognosis, since not infrequently fatal cases of nephritis occur with little or no evidence of it, and, on the other hand, cases presenting most extensive dropsy not uncommonly terminate in complete recovery. The extent of the dropsy is not in proportion to the severity of the inflammation of the kidney, but is believed to be partly due to injury inflicted by the toxins upon the blood and lymph vessels.

Idiopathic Edema.—Attention should be directed to the fact that edema may occur in infants quite apart from disease of the kidneys; this form is most frequently associated with gastroenteritis. I have observed this condition in children suffering from acute enteritis who were being fed exclusively upon beef broth, meat juice, and whiskey, and I have seen the edema quickly disappear when these foods were changed for a modified milk formula. Idiopathic edema is not in any way related to nephritis, and is mentioned here simply to prevent mistakes in diagnosis.

Uremia.—Headache, nausea, and vomiting are the early symptoms which mark the onset of this intoxication. As the toxemia increases, the headache becomes more severe, the stomach more irritable, and diarrhea may occur. Associated with these symptoms there may be a high-tension pulse, muscular twitchings, drowsiness, and vertigo; later, convulsions and coma may occur. Coma is the most unfavorable of these symptoms; persistent vomiting associated with severe headache and disturbances of vision are also alarming symptoms. Since single convulsions not infrequently occur in patients who make a rapid and satisfactory recovery, this symptom cannot therefore be depended upon in making a prognosis.

Urine.—The most important signs of acute nephritis are furnished by an examination of the urine. Albumin is usually abundant, values of over 1 per cent. by weight being not infrequent. The urine in such cases becomes dense with albumin on boiling or by the addition of cold nitric acid. In rare instances only a slight ring of albumin may be present. Casts are of more importance from the standpoint of diagnosis than is

albumin. In all cases of acute nephritis they are found in considerable numbers. Hyalin, granular, blood, and epithelial casts may be distributed through the same field. It should, however, especially be noted that a small amount of albumin and a few hyalin casts, without other signs or symptoms, are not sufficient to make a diagnosis of nephritis; these findings, as already stated, occur in orthostatic and toxic albuminurias, unassociated with inflammatory changes in the kidneys. The urine in acute nephritis, in addition to an abundance of epithelial, granular, and blood casts, contains leukocytes, renal epithelium in various stages of degeneration, microorganisms, and blood corpuscles. A few red blood corpuscles, discovered microscopically, may have little prognostic import, but the presence of blood in marked quantities is an unfavorable sign and usually indicates a severe form of nephritis. But in cases with marked oliguria the hemorrhage may somewhat relieve the congested kidney. The prognostic importance, however, of blood in the urine depends upon its association with albumin and casts. In certain hemorrhagic diseases, such as scurvy, the presence of blood may have little prognostic significance. In acute nephritis, urea, sodium chlorid, and phosphoric acid are diminished. The retention of urea is an especially valuable indication of defective elimination. The amount of uric acid is unchanged, and the aloxuric bases are increased. The urine is nearly always diminished in quantity, is concentrated, reddish brown, or smoky, and has a high specific gravity, 1,030. Occasionally acute nephritis produces complete suppression of urine; this is an ominous symptom, frequently followed by convulsions, coma and death.

Other Symptoms.—In this connection it should be remembered that renal suppression may occur, lasting over twelve or even twenty-four hours, in young children who have no other sign of kidney disease; complete restoration of the kidney function quickly follows in these cases of simple anuria, and they are mentioned here to emphasize the fact that this symptom is of serious import only when it is associated with the other signs and symptoms of acute nephritis.

Fever is an almost constant symptom. A rise in temperature during apparent convalescence from scarlet fever, diphtheria, and other acute infectious diseases should suggest nephritis and lead to an examination of the urine. The temperature commonly runs between 99° and 103° F. A sudden rise during the course of the disease is a bad indication.

A marked secondary anemia rapidly develops, and if the disease passes into the subacute or chronic stage the anemia may be an important symptom in directing attention to the patient's condition.

**Complications.**—The most frequent complications are pleuritis, endocarditis, pericarditis, bronchitis, pneumonia, meningitis, and edema of the larynx.

**Course and Termination.**—Acute primary nephritis occurring in young children is a very fatal disease; in older children recovery is the rule. If these cases can be tided over the first week, a satisfactory recovery may be expected. In scarlatinal and other postinfectious forms recovery is

the rule, but death may occur. It is my experience that the great majority of these cases have little tendency to become chronic. The nephritis of childhood is a disease which has a tendency to spontaneous and complete recovery. If the patient lives, the kidney, in the vast majority of instances, is restored to a normal condition. The fact, however, that chronic nephritis may, in a small percentage of these cases, follow the acute form should make the physician ever careful to see that the recovery from acute nephritis is complete. A pseudo recovery may mislead the physician into withdrawing the restrictions as to diet and exercise which are necessary to complete recovery. The ordinary course of a case of acute nephritis is from three to six weeks, but thereafter the urine should be examined at intervals of five or six weeks to make sure that the recovery is complete.

**Prophylaxis.**—In the treatment of the acute infectious diseases, which are so commonly followed by acute nephritis, the physician should ever keep in mind the prophylactic treatment of this condition. This especially applies to scarlet fever, diphtheria, and severe febrile attacks of acute influenza. In all of these conditions the patient should be confined to bed, and his body kept at rather an even temperature. The diet should be such as to throw little work on the kidney and yet supply nutritional demands. Milk, cereals, fruit juices, and alkaline waters are especially indicated. When vegetables are allowed in the convalescence from these diseases, care should be exercised to exclude rhubarb, tomatoes, and asparagus. In all the acute infections the bowels should be kept open by saline or other cathartics, so that the kidney may not be further irritated by the elimination of intestinal toxins. Warm baths may also be of service in promoting elimination through the skin, and thus diminishing the work which the kidney is called upon to do. The specific treatment of various diseases, such as diphtheria by antitoxin, malaria by quinin, and syphilis by mercury, diminishes the dangers of nephritis in these diseases. Throughout the treatment of all acute diseases, which may be followed by nephritis, the urine should be frequently examined, so that the nephritis may be recognized early and checked by appropriate treatment.

**Treatment.**—General Treatment.—The first object to be sought is the reëstablishment of the kidney secretion, if this function has been materially interfered with. To accomplish this the kidneys must be rested by withholding food, giving water and calling upon the bowels and skin to vicariously do a portion of the work of these diseased organs. Water is the greatest of all diuretics and is indicated at all times in all forms of nephritis. In beginning the treatment a saline cathartic should be prescribed, preferably the sulphate of magnesia; this should be given in suitable doses every three or four hours until free catharsis has been established, and during this time little water and no food should be taken. Sulphate of magnesia, as a rule, produces less nausea, acts more quickly upon the bowels, and eliminates more fluid than any other cathartic. In infants and young children milk of magnesia, Rochelle salts, or a solution of citrate of magnesia

may be used. After the bowels have once been moved, other saline cathartics may be substituted, such as sulphate of soda, phosphate of soda, or one of the alkaline cathartic mineral waters. Later in the disease, when the acute symptoms are under control, vegetable cathartics, such as cascara and compound licorice powder, may be used if the patient finds them more agreeable, but throughout the whole course of the treatment, until actual convalescence has been established, cathartic medication is, as a rule, necessary. In laying stress upon the cathartic treatment of acute nephritis it is important to call attention to the fact that, while in the beginning it must be very active, in the later treatment of the disease care should be taken not to weaken the patient or aggravate his anemia by unnecessary catharsis. In all severe cases, in addition to early cathartic medication, it is advisable to at once begin to stimulate the skin to increased action. This may be done by wrapping the patient in blankets wrung out of hot water (temperature 110° F.), and surrounding him with hot-water bottles, and then covering him with warm, dry blankets; this will produce copious perspiration. If the patient shows no marked depressing effects he should remain in this hot pack for ten or fifteen minutes, after which he may be wiped dry and covered with a warm, dry blanket. The copious sweating which follows these measures is even of greater value in the relief of profound uremic symptoms than is the cathartic medication. Hot packs are especially valuable, however, when nausea and vomiting prevent the cathartic treatment above outlined. In such cases the packs may be supplemented by rectal injections of from a pint to a quart of normal saline solution at a temperature of 105° F., or the same solution may be used by hypodermoclysis, 6 to 12 ounces at a dose. The following formula is recommended by Martin H. Fischer: "Sodium chlorid, 14 grams; sodium carbonate (crystallized), 15 to 30 grams (not bicarbonate); water, 1 liter. This solution is not suitable for subcutaneous injection, but it may be given per rectum by the drop method at a temperature not below 105° F. In urgent cases it may be injected intravenously. For intravenous use it must be sterile. In preparing it, the sterile sodium chlorid solution should be made first, and as this is cooling the sodium carbonate should be added, as heating the latter drives off the $CO_2$. After the formula has been given once by rectum or intravenously it is well to wait three hours before repeating the dose. It takes a little time to get the full effect of the salt and alkali on the kidney. As the kidney function returns, the concentration of the alkali and the salt, in subsequent injections, may be progressively reduced, and finally a simple 0.9 per cent. sodium chlorid solution by rectum, or water salt and alkali by mouth will suffice."

The above formula is the adult dose and is therefore to be diminished proportionately to the age of the child. At the age of five, one-fourth, and at ten, one-half of this dose should be given. Saline injections may be alternated with the hot packs at three or four-hour intervals, until the nausea and vomiting have sufficiently subsided to permit cathartic medication. It should, however, be remembered that, while hot packs are life-

saving measures in the early treatment of these severe cases, they are also very depressing, and are to be discontinued as soon as the uremic symptoms have disappeared and the kidney secretion is again fairly well established. It is rarely necessary to continue the hot packs for longer than three or four days, and in the mild cases it may not be necessary to use them at all. The hot pack is also indicated and may be of great value in cases where there is considerable dropsy. In these cases it is to be administered but once in the twenty-four hours, and its effects are to be carefully studied; if the patient is feeble and the pack produces great prostration, it may do more harm than good. Dry cupping, hot fomentations, electric heaters, and hot-water bottles applied to the lumbar region may not only relieve pain but may also increase the flow of urine by relieving congestion of the kidneys.

In very severe forms of acute nephritis with sudden onset, which are comparatively rare, the intoxication may be so profound and the patient's life may be in such imminent danger that even more radical measures than those above outlined may be indicated to control the uremia and re-establish the kidney secretion. In these violent forms morphin hypodermically, 1/10 to 1/50 of a grain, depending upon the age of the child, may be necessary to control convulsions and vomiting; venesection should then be resorted to, half a pint to a pint of blood being removed. Following venesection, sterile normal salt solution, in quantities equal to the amount of blood removed, should be given by subcutaneous injection, or Fischer's alkaline solution above noted should be given intravenously. Fischer and others have reported very remarkable curative results in many severe cases of uremia from the use of this alkaline solution administered in the manner previously described. It is especially indicated in uremia and severe acidosis. When given intravenously it acts almost specifically in controlling these symptom groups. The collapse of the veins, which occurs in these conditions at times, makes the intravenous injection a very difficult operation.

*Rest in bed* is a necessary part of the treatment of acute nephritis and should be continued until albumin and casts have disappeared. If the heart be weak or the anemia pronounced, albumin may recur in the urine after the patient is allowed to sit up or be upon his feet. In such cases it is necessary to have the child remain in bed until these symptoms are controlled by appropriate treatment. In some instances, where the albuminuria is prolonged, the rest-in-bed treatment may become so irksome to nervous children, especially in hot weather, that it may be necessary to place them in reclining chairs, and shift them from beds to lounges, as the judgment of the physician may direct. This treatment may necessitate confinement to bed for five or six weeks; in the average case, however, this period is much shorter.

Dietetic Treatment.—As above noted, food should be abstained from until the bowels have been acted upon and the nausea and vomiting controlled. Following this, milk should be the chief article of diet. Great

care should be taken at all times not to overload the stomach, or to give food at too frequent intervals. In the early treatment of acute cases small quantities of milk, 4 to 6 ounces, every five or six hours, are all that are necessary. After a few days the quantity of milk may be increased, but the intervals between the feedings should be maintained. If the child dislikes plain milk, then the milk may be flavored or disguised, or one of the proprietary milk foods substituted. The following are recommended: milk with cereal gruels, milk flavored with chocolate, ice cream made from clean, but not rich, milk, milk soups, flavored in various ways, "malt soups," buttermilk, malted milk, condensed milk, and other proprietary milk foods. In this list an easily digested milk food may be found, which the child can be induced to take, that will serve nutritional purposes and remain the basis of the child's diet throughout the disease. A little later, as the kidney secretion becomes better established, fruit juices, bread and cereals may be added to the diet. Orange juice and lemonade are especially grateful. As convalescence approaches, potatoes, and all fresh vegetables, except onions, rhubarb, asparagus, and tomatoes, may be added to the diet; these latter are contraindicated until the child is entirely well. Small quantities of meat and cooked eggs may be given during early convalescence, but are perhaps better let alone until the urine is free from albumin.

Fresh Air.—While the temperature of the sick room should be kept uniformly in the neighborhood of 70° F., the air must always be fresh. Fresh air and iron are the most important agents we have in combating the anemia which is so constant in this disease; the latter is of special value during convalescence. Diuretics are for the most part contraindicated. During the early stages of acute nephritis, when the kidney is sharply inflamed, its secreting structures cannot be stimulated to increased activity by such diuretics as the potash preparations or by remedies such as digitalis that increase the blood pressure. In the later stages of nephritis, however, when the inflammation of the kidney has largely subsided and the heart is weak and rapid in its action, digitalis may be of some value; its action, however, upon the kidney secretion as well as upon the heart should be carefully watched. Some alkaline water or plain water is, as a rule, the only diuretic needed in these cases, and after the nausea and vomiting have subsided the child should be urged to drink water. The nervous or uremic symptoms often demand symptomatic treatment in addition to the hydrotherapy, catharsis, and diet above recommended. Chloroform, morphin, chloral, and bromids may be necessary to control convulsions, and bromid of potash and veronal may be necessary to produce sleep and relieve nervous irritation. I have been much impressed with the importance of avoiding excessive medication in these cases. Any medicine that irritates the stomach or interferes with the appetite will do more harm than good. The treatment in the vast majority of cases is confined to diet, hydrotherapy, catharsis, rest in bed, and fresh air, care at all times being taken to keep the digestive organs in good condition.

## CHAPTER LXXV

### CHRONIC NEPHRITIS AND OTHER DISEASES OF THE KIDNEYS

### CHRONIC NEPHRITIS

Chronic nephritis is comparatively rare in childhood, notwithstanding the fact that this is the period of life in which acute nephritis is most common. Chronic Bright's disease in the adult is not commonly the sequel of acute Bright's disease; in most instances it develops insidiously, and the clinical history fails to trace the beginning of the chronic disease to a previous attack of acute nephritis, which, through neglect, was allowed to develop into the chronic form. In the child, however, this sequence is noted; but, as previously said, the tendency of acute nephritis in childhood is to complete recovery. We do, however, occasionally have the same forms of chronic nephritis in children which we have in the adult, and the symptomatology, pathology and treatment of these chronic forms are practically the same at all ages.

*Chronic diffuse non-indurative nephritis* (chronic parenchymatous nephritis) may follow acute nephritis produced by scarlet fever, influenza, syphilis, and, less rarely, other causes. This form occupies an intermediary position between acute nephritis and chronic indurative nephritis; all three may be stages of the same pathological process. In acute and chronic diffuse nephritis the secreting structures are especially involved in the inflammation. In the indurative form the interstitial tissue is also involved. Amyloid disease is a degenerative process, which may rarely be engrafted upon the chronic nephritis of childhood. Marked amyloid degeneration in the child, as in the adult, is usually associated with tuberculosis, syphilis, suppurative processes, chronic malaria, rickets, and diseases which produce cachexia. Amyloid changes are not confined to the kidneys; other organs, such as the liver, spleen, intestines and suprarenal glands, are also involved.

**Symptomatology.**—The symptoms of chronic nephritis in childhood are the same as the symptoms of chronic nephritis in the adult, except that the disease is not, as a rule, so insidious. When albumin and casts are discovered for the first time in the urine, it is important to determine whether the condition is a chronic one, and, if so, of how long standing. If the disease has lasted for a year it has passed beyond the acute stage, and it is not only unwise, but positively injurious, to subject a patient of this kind to the dietetic and "rest-in-bed" treatment recommended for acute nephritis; the differential diagnosis between acute and chronic nephritis is, therefore, most important. Chronic nephritis differs from acute nephritis in that it is an afebrile disease, and the casts which are found in the urine, while they are largely granular and epithelial, contain large numbers of fat granules, both in and out of the epithelial cells, and free fat drops are commonly seen. Red blood cells, if present at all, are

scarce; albuminuric retinitis is more common; eccentric hypertrophy of the heart with dilatation usually exists. Anemia is more marked, and the whole appearance of the disease is that of a chronic rather than an acute process. Confusion may arise in the differential diagnosis of chronic from acute nephritis when the patient is seen for the first time with an acute exacerbation of a chronic Bright's disease. In such instances the urine has the high specific gravity, the dark color, the excessive quantities of albumin, and the microscopic findings, including red blood cells and blood casts, seen in acute nephritis.

**Treatment.**—The treatment of all acute exacerbations of a chronic nephritis should be the same as that of acute nephritis; the milk diet and rest-in-bed treatment are necessary in these cases until the acute symptoms are controlled. The treatment of chronic nephritis is, however, very different from that of the acute form. These patients should be out of bed, and, if the climate is suitable, in the open air. If the patient can take advantage of climatic treatment he should live the year around in dry, warm, bracing and equable climates; to do this he must necessarily travel with the seasons. Muscular exercise to the point of physical fatigue is to be carefully avoided.

The DIET is also very different from that of acute nephritis. While milk, buttermilk and all the milk foods recommended in the acute disease should form the basis of the diet, yet these patients live much longer and are capable of accomplishing much more if they are given a very general diet. Fruits, cereals, vegetables and albumin should form part of their diet. Albumin is not only permissible, but is really a very valuable food in chronic nephritis. It may be given in the form of cooked (never raw) eggs, fish, chicken, mutton, and even beef in moderate quantity to suit the individual case. Albumin in some form should be given once a day, but care should be taken that the patient does not overeat; that is to say, that the number of calories in the food taken should not exceed that required by a normal individual of the same weight. These patients should avoid alcohol, radishes, asparagus, onions, tomatoes, rhubarb, and all pickled and smoked foods. Water is never contraindicated, even when the urine is scant and the dropsy is marked. In the great majority of cases the kidney secretion is comparatively free, and dropsy is either not present at all, or but slightly marked. In these cases ordinary water, or certain alkaline and lithia waters, such as Poland and Waukesha, should be given in large quantities.

Edebohls' operation of splitting the capsule of the kidney, while not curative, prolongs the life and adds greatly to the comfort of the patient.

## CYSTOPYELITIS

This condition is a purulent infection of the urinary bladder and pelvis of the kidney; it is peculiar to infants and young children. By American and English writers it is commonly described under the term

Pyelitis; by the Germans it is spoken of as Cystitis. It is probable that in the vast majority of cases the bladder is primarily infected and the disease spreads secondarily to the pelvis of the kidney, in some instances involving the kidney structures (pyelonephritis). In a small minority of cases, however, it is known that the infection begins in the kidney and subsequently affects the bladder, so that perhaps the term cystopyelitis most accurately describes this condition.

**Etiology.**—Age and sex are important predisposing factors. It may occur in the first weeks of life, but is comparatively rare before the third month. From this time to the eighteenth month it is common, and thereafter diminishes in frequency, up to the sixth year of life. After this period cystitis in the child is very similar in its etiology and course to cystitis in the adult. The predisposition of females is shown by the fact that only about 10 per cent. of these cases occur in males. Enteritis is the most common predisposing cause. It may also follow influenza, scarlet fever, diphtheria, typhoid fever, gonorrhea and other forms of vaginitis and urethritis. Stone in the kidney or bladder, congenital malformations of the urinary organs, foreign bodies, irritation of the urinary organs by toxins, hyperemia of these organs from cold or other causes, and retention of urine, are spoken of by various writers as predisposing causes.

Exciting Causes.—The colon bacillus is the common exciting cause. This fact was pointed out by Escherich in 1894; he reported 60 cases, in 58 of which this bacillus was found, either in pure or mixed culture. These findings have been confirmed by all subsequent observers. It may therefore be definitely stated that the ordinary cystopyelitis of infancy is due to the bacillus coli communis. Pfaundler further confirmed this etiological relationship by demonstrating an agglutination reaction of these bacilli with the blood serum of a patient sick with this disease. Other microorganisms, however, may produce a purulent cystopyelitis. Among these the following may be mentioned: bacillus proteus vulgaris, bacillus lactis aerogenes, staphylococci, streptococci, gonococci, and typhoid, tubercle, pyocyaneus, and diphtheria bacilli.

Infection in the vast majority of cases occurs from below upward through the urethral canal. In these cases cystitis is probably the primary, and pyelitis the secondary, lesion; this is especially true of infection by the colon bacillus. Infection may occasionally occur through the blood stream, primarily affecting the kidney. The infecting microorganisms may also migrate directly through intervening tissues from the intestinal canal to the urinary organs. The two latter modes of infection, however, are comparatively rare, so that from a clinical standpoint it is well to recognize the fact, not only that the vast majority of these cases are caused by colon bacilli, but that these infecting organisms commonly find an entrance through the urethral canal to the urinary bladder and later to the kidney. With these facts in mind one can understand why this disease occurs so frequently in young female infants whose urethral canals are exposed to

contamination from fecal discharges, and also why this disease is so commonly the sequel of enteritis.

**Symptomatology.**—General Symptoms.—*Local symptoms,* such as pain and tenderness over the bladder and frequent and painful urination, may lead to the discovery of this condition through an examination of the urine. In the majority of cases local symptoms on the part of the genito-urinary organs are either not present at all, or are so slight as to be overlooked. In this fact lies the difficulty of diagnosis, or rather, one should say, it explains why this condition is so commonly overlooked. The diagnosis of these cases is simple enough where the symptoms are such as to cause the physician to make a urine examination, but since routine examinations of the urine of infants are not commonly made in private practice he depends upon the constitutional, rather than the local, symptoms to direct his attention to the location of this infection. An unexplained fever is the most notable and constant of these constitutional symptoms. It is commonly associated with more or less gastrointestinal disturbance and frequently with the following symptoms: restlessness, nervous irritability, anorexia, nausea, abdominal colic, pain and tenderness over the bladder, and a more or less marked and progressive pallor. If physicians would examine the urine of every infant suffering from a fever the cause of which was not clearly defined, but few of these cases would be overlooked.

The *fever* in cystopyelitis is usually continuous, showing marked remissions. It may, however, be intermittent or septic in character, rising to 104° or 105°F., and falling, within twenty-four hours, below normal. In acute cases the fever is important, both from the standpoint of diagnosis and prognosis; the bad cases are marked by high and irregular fever and a cessation of the febrile process usually means approaching convalescence. In mild cases the febrile reaction may be slight. Chronic cases may continue for an indefinite period with little or no elevation of temperature.

In very severe cases the *clinical picture* may be even more obscured and attention may be directed away from the urinary tract by symptoms resembling typhoid fever, pneumonia, severe intestinal intoxication or meningitis. That this disease does occasionally present the appearance of the above-named infections is a fact which should be kept in mind. Mistakes in diagnosis can be prevented only by systematic urine examinations in all infectious processes characterized by pronounced constitutional symptoms.

In chronic cases this disease may be complicated by the coexistence of the symptom-complex elsewhere described as *recurrent vomiting.* I have seen cases of this kind, such as are reported by Porter and Fleischner.

A tender *tumor* in the kidney region may sometimes be found by palpation. This, as a rule, indicates a pyelonephritis or a pyelitis, in which the pus is distending the pelvis of the kidney. In these cases the fever curve is high and intermittent; that is to say, markedly septic in character, and the other constitutional symptoms are much aggravated. Convulsions and other uremic symptoms may intervene in the fatal cases.

Urine.—The diagnosis is made by the urine findings. Pus cells in great numbers, as well as renal (caudate) and bladder (squamous) epithelium, are found. A small quantity of albumin and occasional casts, hyalin and granular, are present in many of the cases. The specific microorganisms causing the disease may be discovered if the proper technique is used.

The urine in the majority of these cases is acid in reaction; this is true in all cases produced by the colon bacillus, and in the extremely rare cases produced by the tubercle bacillus. It is alkaline in reaction when the disease is produced by septic cocci. In the more severe cases where the kidney structure is involved the filtered urine contains much albumin, and a microscopic examination shows many epithelial, granular and blood casts; blood corpuscles may also be found.

In chronic cases an X-ray picture may be of value in determining the size of the kidney, and the presence or absence of a kidney stone.

**Course and Termination.**—A small percentage terminate fatally as a result of kidney involvement or general sepsis. In about 95 per cent. the prognosis, so far as life is concerned, is favorable, and the tendency, as a rule, is to spontaneous and complete recovery. A considerable number of the cases which ultimately recover pass from the acute to the subacute form of this disease and continue for many months or even years.

**Prognosis.**—The prognosis depends largely upon the character of the initial infection. The colon cases are the most favorable; these most commonly terminate in recovery in from two to eight weeks. Cases in which septic cocci predominate are more prolonged, more severe and more dangerous. If diphtheria bacilli are found, the disease is even more serious, and the finding of tubercle bacilli usually justifies an unfavorable prognosis. It should be remembered that simple cases, due primarily to colon bacilli, may by neglect or maltreatment be converted into severe and dangerous cases of mixed infection, in which the septic cocci play the most important rôle.

Relapses may occur in cases that have apparently recovered; Abt reports two relapses in twenty cases; one of these had three attacks.

**Prophylaxis.**—The prophylactic treatment consists in keeping the genital organs of the infant clean, by changing the diaper and carefully washing and drying the external genitalia as soon after the discharge of fecal matter as possible, the object being to prevent the colon bacillus from entering the urethral canal. Vaginitis and urethritis in the infant and young child should receive prompt and careful attention.

**Treatment.**—Some of the milder cases require little treatment other than quiet, cleanliness, and careful attention to diet. Enteritis when present should receive attention. Alkaline waters are valuable diuretics. Citrate of potash, recommended by Holt, in 3- to 5-grain doses three times a day, is of value. One tablespoonful of a saturated solution of sodium phosphate in every bottle or glass of milk I have found of great service in chronic cases associated with constipation. Bicarbonate of soda may also be given in the food.

Urotropin is perhaps our most valuable remedy during the acute stage. It may be given three times a day, in from one- to three-grain doses, depending upon the age of the child; it is an effective urinary antiseptic, which materially assists in shortening the course of the disease. In some instances, as Abt has noted, this drug may cause "renal and vesicle irritation"; it is then contraindicated. Salol is a valuable remedy, especially in young infants; it may be given three times a day, in two- to five-grain doses, according to the age of the child, without disturbing the gastro-intestinal tract; it acts as a mild urinary antiseptic. Guaiacol, which is excreted largely through the urine, acts like urotropin and salol as a urinary antiseptic; it is especially valuable in very young infants. It may be given in the form of the carbonate in two- to four-grain doses three times a day, or in liquid form it may perhaps better be administered by inunction. For this purpose one drachm of liquid guaiacol is combined with one ounce of anhydrous lanolin, and one drachm of this ointment is thoroughly rubbed into the skin of the abdomen or axillary regions once a day (see chapter on General Therapeutics).

Both autogenous and stock bacillus coli vaccines have been used very successfully in these cases. This vaccine treatment should, therefore, be tried in cases that fail to respond in a reasonable time to the above-named measures.

Irrigation of the bladder is, as a rule, inadvisable and unnecessary; in refractory cases, however, it may be resorted to. For this purpose a weak solution of nitrate of silver, 1-2,000, or a saturated solution of boracic acid are recommended. High temperatures may be treated by ice-bags to the head and sponging with cold water. In those rare cases where the X-ray locates a stone or other foreign body in the urinary passages surgical intervention is necessary.

## TUMORS OF THE KIDNEY

Sarcoma is by far the most common of kidney tumors. Osler says that nearly all large, solid, abdominal tumors in children are sarcomatous. Birch-Hirschfeld describes these malignant tumors of the kidney under the term embryonal adenosarcomata, and believes that primary carcinomata are relatively rare. From a clinical standpoint further classification than this is unnecessary, since all malignant tumors of the kidney have practically the same etiology, symptomatology, prognosis and treatment. Usually only one kidney is involved, the left more frequently than the right.

**Etiology.**—Little or nothing is known of the causative factors. Chronic irritations, from traumatic causes, are said to precede the formation of tumors in many cases. They have their origin in embryonal tissues, and, as they develop, the kidney structure is gradually destroyed by compression; they push out from the region of the kidney downward and inward, until in time they may fill the entire abdominal cavity. In their early

development the kidney alone may be involved, but late in the disease metastasis may occur, involving the liver, lymph nodes, and other organs; the bladder, however, is rarely affected. These tumors may be present in the new-born; the great majority of them occur during the first five years; they are very rare after the tenth year.

**Symptomatology.**—The tumor is the characteristic symptom; in most instances it develops insidiously. Attention may be called to this condition by the appearance of blood in the urine, but, as a rule, enlargement of the abdomen is the first symptom noticed, and a physical examination then reveals the tumor. In its early stages it may be unilateral, and may be felt protruding from the region of the kidney downward and inward into the abdominal cavity; later it may become so large as to produce great abdominal distention so symmetrical that a physical examination may with difficulty discover which kidney is involved. Small tumors, discovered early, are firm and solid; large ones are soft and doughy to the touch. These, when they have reached a size where they can be easily palpated,

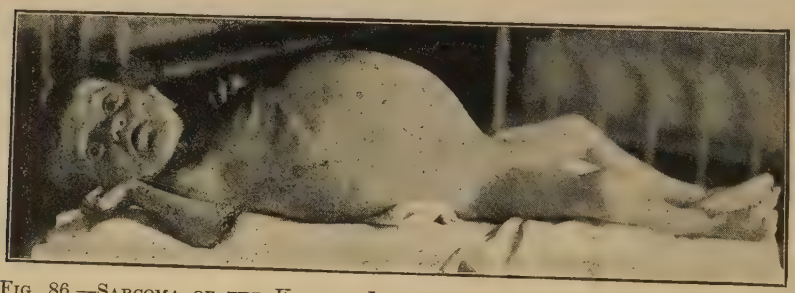


Fig. 86.—Sarcoma of the Kidney; Infant Four Months Old. (J. F. Bell.)

grow rapidly, and should not be mistaken for enlargement of the liver or spleen. Both of these organs, when enlarged, produce a board-like smooth tumor, the edge of which can be sharply outlined by palpation and percussion; this is not true of malignant growths. The location of the distended colon running along and above these tumors may be made out and is of importance from the standpoint of differentiation, particularly from enlargements of the spleen and liver.

Hematuria is a common and early symptom which is said to occur more frequently in carcinoma. The blood may be so abundant as to be made out by the naked eye; in most instances, however, the microscope reveals the hemorrhage. Pain is a common but not a characteristic symptom; it may be severe, but is usually dull, producing discomfort and irritability. As the tumor becomes very large pressure symptoms appear, and displacements of the abdominal viscera are common. In the later stages cachexia, emaciation and loss of strength are pronounced symptoms. Ascites and swelling of adjacent lymph nodes are also present. When both kidneys are involved, uremic symptoms may mark the closing stages of the disease.

**Treatment.**—The complete removal of these tumors by surgical measures is advised in all cases where cachexia and profound nutritional disturbances do not exist; about 70 or 75 per cent. survive the operation; 6 or 7 per cent. of these ultimately recover, and those that do not recover are, for the time being, made more comfortable and life is prolonged. If the operation is made while the tumor is yet small and confined to the kidney, the prognosis is much more favorable. The symptomatic medical treatment of these cases consists in making the patient as comfortable as possible by relieving pain and nervous irritability by the judicious use of phenacetin, aspirin, or some preparation of opium, such as paregoric, codein or morphin.

## HYDRONEPHROSIS

Hydronephrosis in children is a comparatively rare condition. It is commonly congenital, but there is an acquired form, which may occur late in childhood.

**Etiology.**—It is due to obstruction in some part of the urinary canal, commonly the ureters; in rare instances the stenosis may occur lower down in the bladder or urethra. The blocking of the urinary canal may be caused by calculi, inflammatory adhesions, malformations, twisting of the ureters and tumors. These mechanical obstructions impede or prevent the flow of urine through the urinary canal, and this results in an accumulation of fluid in the pelvis of the kidney, which gradually produces a fluctuating tumor. The pressure of this fluid may destroy the kidney substance.

**Symptomatology.**—The characteristic symptom is a fluctuating tumor which may be large enough to extend downward and inward from the region of the kidney well into the abdominal cavity. Aspiration of this tumor obtains an albuminous fluid, containing urates, urea, and epithelium. Hydronephrosis is usually unilateral; when bilateral, it early terminates fatally.

**Treatment.**—When hydronephrosis is unilateral, and the urine discharged shows that the other kidney is normal, radical surgical measures for the removal of the diseased kidney are justifiable. Such cases are very rare and are more commonly found in the acquired form in older children.

## CYSTIC DEGENERATION OF THE KIDNEY

Cystic degeneration of the kidney is congenital and almost always bilateral. By the development of cysts in the kidney structure the functional efficiency of these organs is destroyed, and the infant dies from uremia. This condition is fortunately rare, and is of little interest except from the standpoint of diagnosis. Congenital cystic kidneys are palpable at birth.

## TUBERCULOSIS OF THE KIDNEY

Tuberculosis of the kidney is almost always a secondary condition. The diagnosis is made by the symptoms and signs of tuberculosis elsewhere in the body and by the finding of tubercle bacilli in the urine. The urines of these cases usually contain red blood cells, albumin and pus. Progressive anemia, loss of weight and strength, an irregular temperature curve, and other symptoms of tuberculosis are usually present.

## PERINEPHRITIS

Perinephritis is an inflammation of the tissue in which the kidney is imbedded; it is a rare condition, commonly terminating in abscess. The disease is secondary to tuberculosis, or pyogenic infections in the kidney or elsewhere in the body. Traumatism is classed as an important etiological factor. It is important to keep in mind that perinephritic abscesses, although rare, may occur, and that the pus may burrow downward, forming a fluctuating mass beneath the liver or the spleen. In these cases there is tenderness on pressure in the lumbar region, and, on deep pressure, bimanual examination may reveal a soft tumor. Chills, fever, and the symptoms of septicopyemia are present. The treatment of this condition is surgical.

## DISLOCATION OF THE KIDNEY

Both the kidney and suprarenal glands are relatively large in infancy; at birth the suprarenals are nearly one-third the size of the kidneys. The kidney itself is 1/100 of the body weight, while in the adult it is about 1/230, so that at birth it is relatively more than twice as large as in the adult. The lobulated form of the fetal kidney persists for a short time after birth. Notwithstanding the relatively large size of the kidney at this period of life, it cannot be palpated, nor can it be readily located by percussion. A kidney, therefore, that can be palpated at or shortly after birth may, as a rule, be classed as a congenital dislocation. There are two types of this deformity: the floating kidney, which is rare in infancy, and dystopia or downward displacement of the kidney, which is still more rare. In the latter condition the kidney is not more movable than normal, but is found well down in the abdomen. This displaced kidney is commonly lobulated and its ureter is short.

## CHAPTER LXXVI

### DISEASES OF THE GENITAL ORGANS

## GONORRHEAL VULVOVAGINITIS

Gonorrheal vulvovaginitis in infants and young children differs from the gonorrhea of the older child and adult, in that it is rarely a venereal disease. It is caused by the accidental inoculation of the vulvovaginal canal with the gonococcus of Neisser. It is confined exclusively to female infants and young female children. The corresponding condition, gonorrheal urethritis, is very rare in the male infant and child. It occurs in extensive and at times almost uncontrollable epidemics in institutions for infants and young children. In this tendency to spread in epidemic form without sexual contact, it is wholly unlike gonorrhea in the adult; it also manifests itself in a less virulent form, and has fewer complications. In recent years it has become much more prevalent, so that at the present time it can usually be discovered in all large institutions caring for female children. Holt finds that 5 per cent. of cases applying for admission to the Babies' Hospital in New York have this disease. It is also not uncommonly seen in private practice. Holt graphically describes the difficulties of combating it in hospital practice, and notes the fact that 273 cases occurred within five years in the Babies' Hospital, in spite of "the united efforts of the physicians and superintendents in quarantine and disinfection."

**Etiology.**—Institutional epidemics can usually be traced to the admission of a case of this disease into a ward containing other children. It is then rapidly spread by means of bathtubs, diapers, underclothing, bedlinen, thermometers, and other things that may carry the contagion from the infected child to the genital tracts of other children. Holt has especially dwelt upon the fact that the nurses' hands may frequently carry this contagion. It is the testimony of all physicians who have studied the spread of these epidemics in very young children that in some indirect way the genital tracts are readily inoculated with the contagion when it exists in the ward, and also that it is almost impossible to prevent the spread of this disease if the infected child is allowed to remain in the ward, or to associate in any way with well children. It may occur in the new-born; in such cases the vaginal canal of the infant is inoculated during birth by the vaginal discharges of the mother. The great majority of cases occur between the third and fifth year of life. It will thus be seen that the vaginal canal of the young child is remarkably susceptible to this contagion, and it is believed by many writers that scarlet fever (Alice Hamilton), measles, and possibly other acute infections render them even more susceptible. The writer in 1906 reported an epidemic occurring in a diphtheria ward, but he attributed the spread of the disease to overcrowd-

ing rather than to an increased predisposition produced by diphtheria. Gonorrheal vaginitis occurring in older children, from seven to twelve years of age, may be due to sexual contact, or it may result from the child sleeping in the same bed with an adult who has this disease. These cases, both in their etiology and clinical course, more closely resemble the disease as it occurs in the adult.

**Symptomatology.**—In the great majority of cases the disease is discovered accidentally, there being no subjective symptoms to call attention to disease of the genital tract. The vaginal discharge is the all-important sign. It may be so slight as to be almost unrecognizable, or so copious as to stain the napkins, underclothing or bed-linen. It commonly has the appearance of mucopus; in some cases it may be glairy and tenacious; in others it may be thin, yellowish, greenish, and tinged with blood. In aggravated cases small ulcerations may occur. The experience of recent years has demonstrated that the vast majority, perhaps 95 per cent., of severe cases of vaginitis occurring in young children is due to the gonococcus, or at least one may say that in such cases a microorganism, which cannot be differentiated from the gonococcus of Neisser, is found. In practically all of these cases, however, the gonococcus is associated with other microorganisms, such as are found in cases of so-called simple vaginitis; among these may be mentioned streptococci, staphylococci, colon bacilli and pseudodiphtheritic bacilli. The ultimate diagnosis in these cases is made by the demonstration of gonococci in the vaginal discharges. This demonstration is commonly made by finding the gonococci within the pus cells. The failure of these cocci to decolorize under Gram's method of staining is considered characteristic. In long-standing cases, as Heimann and others have pointed out, gonococci may be demonstrated only by culture methods.

In mild cases there may be very little swelling of the labia and vagina, but in others these parts may be red, swollen and much irritated. In some instances the child complains of discomfort, and painful urination may indicate that the urethra is involved. The inflammation may be limited to the vagina in the early stages, but in the great majority of cases it is probable, as Koplik insists, that the cervix uteri is also inflamed, and this is especially true of children over six years of age. In infants and very young children the disease is probably more frequently confined to the vagina. In children over seven years of age the inflammation is usually more severe, the secretion more copious, the parts more swollen and the Fallopian tubes, as well as the uterus, more likely to be involved; these are the cases which may possibly produce sterility in after life. Suppuration of the inguinal glands may rarely occur in older children. This condition is, for the most part, an afebrile disease, but in some cases during the most acute stage the temperature rises to 101°F. In long-continued cases nutrition may suffer; this is probably due to the confinement, and lack of fresh air and exercise, which the treatment entails.

**Complications.**—Complications are comparatively rare. I treated a

series of 40 cases in the Cincinnati Hospital without a single complication. During this time, however, three cases of gonorrheal conjunctivitis were admitted; in all of them there was a coexistent vaginitis. The literature of this subject shows that the following complications may occur: conjunctivitis, arthritis, endo- and pericarditis, stomatitis, peritonitis, cystitis and proctitis; of these, conjunctivitis is by far the most common. Holt reports 26 cases of acute gonococcus arthritis, 19 of which were in male infants, and only three presented at any time any other evidences of gonorrheal infection; two were associated with vaginitis.

**Prognosis.**—Nearly all of these cases finally get well; very few of them terminate fatally. The course of this disease is, as a rule, tedious; it usually continues for from one to four months, and may last for years; relapses are common in cases apparently cured. Occasionally an eye may be lost, and sterility may possibly result in older children.

**Prophylaxis.**—The prophylactic measures necessary to prevent the spread of this disease in institutions which care for female children may be outlined as follows: All female children applying for admission should be carefully examined, and if there be the slightest evidence of vulvovaginal irritation or catarrh they should be kept carefully isolated until repeated microscopical examinations of their vaginal secretions have demonstrated the presence or absence of the gonococcus. The fact that the vast majority of these cases of vaginal catarrh sooner or later show the presence of the gonococcus has caused most clinicians, who have had experience with this disease in institutions, to exclude all cases of this kind from children's wards, even though the gonococcus cannot be demonstrated. This latter plan is perhaps the safest rule to follow, since the non-gonorrheal cases are also infectious. During an epidemic it is wise to set apart a ward where all infected cases may be at once isolated from those not having the disease. Infected children should wear diapers holding pads of gauze covering the genitalia, and these pads should be changed frequently, and destroyed by burning. All clothing, linen, etc., should be soaked in bichlorid solution before they are sent to the wash. Nurses should be carefully instructed as to the danger of carrying the infection on their hands and thus producing conjunctivitis, or reinoculating the vaginal canals of convalescent children. To avoid these dangers the nurse should thoroughly cleanse and disinfect her hands after giving the treatment or handling infected clothing. The ward bathtub should not be used for bathing purposes, and the toilets should be carefully cleansed after each individual use. Each patient should be provided with a separate catheter and other instruments used in the treatment, and following the treatment these should be carefully cleansed and placed in separate bottles containing carbolic acid solution and labeled with the child's name.

**Treatment.**—The most important part of the treatment is the thorough douching twice a day of the vaginal canal with large quantities of some non-irritating alkaline antiseptic. In my experience the degree of success will depend not alone upon the care which is exercised in avoiding injury

to the inflamed parts, but also on the character of the instrument used for carrying the antiseptic solution into the vagina. The small female catheter, which is almost universally recommended, is not at all suited for this purpose, since the blunt end of the catheter pushes the discharge ahead of it to a point high up in the vagina, quite out of reach of the antiseptic solution which passes through the eye of the catheter. After a long and unsuccessful use of this and other instruments, I conceived the idea of having this same small-sized female catheter perforated at the end and an additional hole made in the opposite side, so that there would be three openings through which the antiseptic fluid might escape in irrigating the vagina. The hole in the end of the catheter is of the greatest importance, as it allows thorough irrigation of the cervix of the uterus and the vault of the vagina. It is also important that the catheter used should be small enough to insert with ease into the vagina, and the nurse should be instructed to exercise great care in its use, so that the slightest trauma to the parts may be prevented. In 1906 I recorded my success with this method of treatment, and since that time I have been more and more impressed with the fact that attention to details, as above outlined, is absolutely necessary to success.

In my experience the best irrigating solution is normal salt solution containing 5 to 10 per cent. of boracic acid; of this two or three quarts are to be used night and morning, to be followed by the injection through the same catheter of three ounces of a 1 per cent. solution of nitrate of silver. As the inflammation subsides the strength of the nitrate of silver solution may be increased to 2 or 3 per cent. This is to be continued for ten days after the discharge has ceased and gonococci are no longer found. The patient should then be transferred to another ward and kept under observation for ten days or two weeks without treatment. If at the end of that time there is no return of the vaginal discharge and no gonococci are found, the patient may be finally discharged.

Other irrigating solutions, such as a weak solution of permanganate of potash, or a saturated solution of boracic acid, have been widely recommended in these cases, and 10 to 40 per cent. solutions of argyrol have also been successfully used, following the antiseptic douche. My experience with the vaginal douche above outlined has been very large, and I have never seen bad results follow its use, and am, therefore, inclined to think its value is so great, and the risk of infecting the cavity of the uterus so slight, that it should unhesitatingly be used in all cases.

Vaccine Treatment.—Investigations have shown that these cases may be benefited by vaccine treatment, but it is advisable in each case to determine the opsonic index of the individual before the treatment is begun, in order that the value of the vaccine injections may be correctly determined. Alice Hamilton made a careful study of this treatment and came to the following conclusion: "Better results are obtained by the use of strains which have been grown for a long period on artificial media than by the use of freshly isolated strains, and there appears to be no advan-

tage in using the patient's own organism. While the inoculation treatment does not produce a marked effect during the first weeks in acute cases, it seems to shorten the later stages; in chronic cases its effects are more evident than in acute. It is desirable, though not absolutely essential, to control the inoculation by the patient's opsonic index."

## SIMPLE VULVOVAGINITIS

Koplik states that this condition is not infrequent, and that the local symptoms are very similar to the gonorrheal form of this disease. The differential diagnosis between the two is made by the presence or absence of the gonococcus in the vaginal discharges. The great majority of other writers have found simple vaginitis in children under six years of age to be a comparatively rare disease. Only about 5 per cent. of the well-marked cases of vaginitis belong to this class; in 95 per cent. the gonococcus is found some time or other in the course of the disease. In children over six years of age, however, a simple, mild, vaginal catarrh is not so infrequent.

**Etiology.**—It is believed, especially in older children, that general malnutrition and the acute infections may predispose to this disease; tuberculosis, anemia, measles, influenza, and scarlet fever may be etiological factors. Among the exciting causes the following have been named: dirt, foreign bodies, pin worms, scabies, masturbation, and traumas from any cause. The inflammation is maintained by streptococci, staphylococci, colon bacilli, pseudodiphtheritic bacilli, and perhaps other microorganisms. One or more of these bacteria may be demonstrated in the vaginal discharge. The diagnosis of simple vaginitis depends not alone upon the presence of a mucopurulent discharge, but upon the failure to demonstrate gonococci in this discharge. The local symptoms differ from those of true gonococcus vaginitis only in the severity of the inflammatory process. The labia and vagina are red and swollen and covered with a mucous or purulent secretion, and the catarrhal process may, in severe cases, involve the cervix. The urethra may also be involved, producing painful micturition.

**Course and Treatment.**—These cases yield more readily to treatment than those of gonococcus vaginitis; they may commonly be brought under control within two or three weeks. The local treatment is the same as that outlined for gonococcus vaginitis. If tuberculosis, anemia, or other forms of malnutrition be present, these conditions should receive appropriate treatment, and ofttimes the cure of the local condition waits upon the cure of the underlying constitutional trouble.

## URETHRITIS IN MALE CHILDREN

Simple urethritis resulting from uncleanliness and infection may occur in young children. The meatus is swollen and inflamed, and on pressure a few drops of pus may exude from the urethra. Urination is more or less

painful. The condition yields readily to treatment. The external parts are to be kept clean, dusted with an antiseptic powder. Salol and bicarbonate of potash should be given internally.

Gonorrheal urethritis occurring in older boys has the same etiology and treatment as in the adult.

## ADHERENT PREPUCE

Adherent prepuce is due to an agglutination of the epithelial layers of the glands and prepuce; it is present in nearly every male infant. The treatment consists in forcibly retracting the prepuce, separating the adhesions, removing the retained smegma and anointing the parts with vaselin. This procedure is to be repeated at intervals of a few days, until there is no longer any tendency to agglutination of the mucous membranes. From time to time throughout infancy and early childhood this process may have to be repeated.

## PHIMOSIS

Phimosis may result from a long and thickened prepuce, from a congenital narrowness of the preputial orifice and from inflammation of the parts (balanitis).

**Symptomatology.**—Phimosis is very common; in many cases the child suffers no inconvenience and symptoms calling attention to the condition may be absent; in others the parts may be inflamed and irritated and the passage of urine causes pain and increased irritation. Where the opening is very small the urine may have difficulty in escaping, and ballooning of the prepuce may occur with urination; in these cases the retention and decomposition of the urine may add to the irritation and produce a balanitis. Phimosis not infrequently induces priapism, and infantile masturbation may result. It is also one of the most common reflex factors of such neurotic disturbances as night terrors, incontinence of urine, general nervous irritability, hysteria and sleeplessness.

**Treatment.**—In mild cases the preputial orifice should be dilated, the foreskin retracted, the agglutinated surfaces of the mucous membranes separated, the smegma carefully removed and the parts anointed with vaselin or some other ointment. This process is to be repeated from time to time until the preputial orifice is fully dilated and the mucous membranes are no longer in a condition of irritation that will result in their agglutination. In cases where this procedure is not followed by success, or in those cases in which the prepuce is very long and the preputial orifice is very narrow, circumcision should be resorted to. Circumcision is very much to be preferred to the dilation treatment if the latter has to be continued over a long period of time.

## PARAPHIMOSIS

Paraphimosis is a complication which sometimes occurs in phimosis when the foreskin, retracted over the glands, becomes strangulated in this

position. It is characterized by marked edema of the strangulated prepuce and congestion of the glands. In some cases this strangulation may be overcome by pressure upon the glands in such a manner as to force it through the constriction. If this manipulation fails the constriction must be relieved by a surgical operation and circumcision may be performed at the same time.

## HYDROCELE

Hydrocele is an accumulation of fluid in the peritoneal sac surrounding the testicle and epididymis. In the congenital variety there is a direct communication between the hydrocele sac and the peritoneal cavity, and the fluid may be pressed upward and made to disappear in the abdominal cavity. In true hydrocele of the tunica vaginalis the upper portion of the hydrocele sac is closed, and the tumor cannot be made to disappear by pressure; in this condition a well-marked oval tumor is present, either on one or both sides of the scrotum. Fluctuation may be made out, and the illumination test may differentiate the testicle from the more translucent hydrocele fluid. In hydrocele of the cord the tumor extends upward and is elongated; an encysted hydrocele of the cord occurs when the hydrocele fluid is held in this position between two constrictions.

**Treatment.**—Hydrocele in infancy usually disappears without treatment. If, however, the condition persists, the fluid may be drawn off by puncturing the sac; should the tumor recur from time to time, the withdrawal of the fluid may be followed by the injection of 1 or 2 drops of tincture of iodin, in the hope that the irritation following this procedure will obliterate the sac. If the congenital form persists, a truss may be worn to facilitate adhesions in the canal which communicates with the abdominal cavity.

## UNDESCENDED TESTICLE

The testicles are usually found in the scrotum at or shortly after birth. In rare instances, however, the testicle does not descend; it may remain in the inguinal canal or in the abdominal cavity. The diagnosis is made by an examination of the parts which results in finding the scrotum empty on one side, rarely on both, and a small tumor, the size of the infantile testicle, in the inguinal canal. Undescended testicle, as a rule, requires no treatment unless it be so caught in the inguinal canal as to be pressed upon and give rise to pain and irritation. Under these conditions surgical treatment may be necessary to remove the testicle from its position in the canal into the abdominal cavity, or, if possible, to its normal position in the scrotum.

## HYPOSPADIAS

Hypospadias is a congenital deformity of the male genital organs, in which the urethra does not extend to its normal orifice in the head of the

penis. It opens on the under surface of this organ at some point between the base of the scrotum and the end of the penis. Beyond the opening the urethra may be represented by an open fissure extending some distance along the inferior surface of the penis. In severe cases the opening may occur in the perineal region, producing a fissure which extends beneath the scrotum, and because of the rudimentary condition of the penis and undescended testicle, which are not infrequently associated with this condition, it may be mistaken for hermaphrodism. The incontinence of urine which occurs in these cases is a source of great annoyance and results in more or less irritation of the parts. In the milder cases surgical intervention may partially overcome this deformity.

## EPISPADIAS

Epispadias is a very rare malformation in which the urethra opens on the upper surface of the penis and beyond this opening a furrow may extend to the glans.

## ENURESIS

Enuresis in children is a symptom usually neurotic in origin. It is not, as a rule, associated with a muscular incompetency of the sphincter vesicae. The cases of incontinence of urine due to malformations and paralysis are not included under this heading. Enuresis, like the other neuroses of childhood, commonly rests upon a tripod of etiological factors, viz.: 1, irritable and unstable nerve centers due to age and heredity; 2, bad blood and consequent malnutrition; 3, reflex irritation. In many cases these three factors coexist. It is not wise, without a most careful examination of the individual case, to assume that any one of these factors is the sole cause of this condition. A rational inquiry into the etiology of a case of enuresis must seek for the presence or absence of each of these factors, and must determine their relative importance in producing this syndrome.

The detrusor muscles of the bladder, which by their contraction expel its contents, and the sphincter muscle, which by its contraction prevents the escape of urine from the bladder, are enervated by sensory and motor nerves from the lumbar cord. The bladder is emptied, or its contents retained, according to the paths through which the nervous impulse from the lumbar center is carried. Another most important fact to bear in mind is that while the urination center in the spinal cord may be excited to discharge its impulses from reflex excitation, it is, to a large extent, in normal children, under the inhibitory control of higher centers, including the voluntary centers in the brain cortex. This inhibitory function of the higher centers exercises a marked control over the discharge of nerve force to the bladder from the urination center in the cord; the act of urination is, for this reason, largely under the control of inhibitory

centers and partly under control of the will. We will to urinate or not to urinate, and the message passes down to the center in the lumbar cord where, by the mechanism just described, the bladder is emptied or its contents retained.

**Etiology.**—The etiological factors of enuresis may also be divided with reference to the manner of their action, into three classes: First, those that act upon the higher centers, diminishing their inhibitory control over the urination center in the lumbar cord; second, those that act directly on this center in the cord, making it more irritable and unstable, and in that way increasing its reflex excitability; third, those that act by reflex irritation indirectly on the spinal centers, touching off the nervous impulses which produce urination.

Predisposing Causes.—*Age* is a most important predisposing factor. In early life the nerve centers are more excitable and reflex phenomena of all kinds are greatly exaggerated, and in addition to this there is a functional immaturity of the centers inhibiting reflex acts. In early infancy inhibition is so feebly developed that we have a normal incontinence of urine. As the infant grows older the mechanism inhibiting reflex acts, becomes better developed, so that by training it may acquire fair control of the bladder during waking hours, about the seventh or eighth month of life; but, during sleep, incontinence of urine may continue, even in the normal child, through the second year; after the third year it should be considered pathological. The delayed development of the mechanism which controls urination, in the great majority of instances, does not persist beyond the seventh year, but enuresis from various causes may continue into adult life.

*Heredity.*—A neurotic inheritance is an important predisposing cause of enuresis. It may manifest itself as a family tendency, and children with enuresis not infrequently have other nervous symptoms.

*Chronic malnutritions* due to tuberculosis, improper feeding, unhygienic surroundings, enteritis, rheumatism, malaria and syphilis are potent factors in producing enuresis. Influenza and other acute infections, by interfering with the child's general nutrition, may prolong or cause a recurrence of enuresis. Children of gouty parents who have inherited a marked uric acid diathesis, not infrequently suffer from this condition. As Williams has noted, a thyroid insufficiency may also produce enuresis.

Exciting Causes.—*Reflex irritation* in some form is such an important exciting cause of enuresis that it can be found in about half of the cases. The reflexes which are most closely associated with enuresis have their origin, as a rule, in genital, vesicle and rectal irritations. The most important reflex causes are phimosis, preputial adhesions, contraction of, or granulations in, the meatus, vaginitis, urethritis, hyperacidity of the urine, an excess of oxalates and urates in the urine, bacteriuria (commonly due to colon bacilli), cystitis, calculi, contracted and intolerant bladder, thread worms, fissure and eczema of the anus, and rectal polypi. Reflex irritations having their origin in diseases of distant organs such as

the throat, nose, eye and intestinal canal, may be associated with enuresis, and the removal of these distant reflex causes of irritation may exercise a favorable influence on the course of the disease.

*Habit.*—It should be remembered that whatever may have been the important etiological factors of enuresis, the condition may continue even after they are apparently removed. The continuance of the enuresis in these cases may in part be due to the spinal irritability which still persists, but it may be due to the habit which has been formed of emptying the bladder when it contains but a small quantity of urine; this habit is apparently engrafted upon the nervous mechanism which controls urination.

Ruhräh offers the following table as a summary of the causes of nocturnal enuresis:

Physiological—Taking too much fluid.

- Eliminative
  - Due to faulty metabolism.
  - Eating too much salt, etc.
  - Due to drugs.
- Urine
  - Hyperacidity.
  - Alkalinity.
  - Bacteriuria.
- Genitourinary organs
  - Inflammations
    - Urethritis.
    - Cystitis.
    - Pyelitis.
  - Malformations.
  - Calculi.
  - Tumors or polypi.
  - Hypertrophy.
- Nervous system
  - Hypertonia or irritability of bladder.
  - Weakness of sphincter.
  - Reflex
    - Balanitis.
    - Vulvovaginitis.
    - Anal fissure.
    - Rectal polypi.
    - Intestinal parasites.
  - Malformation of spinal cord.
  - General irritability.
- General
  - Diabetes mellitus.
  - Diabetes insipidus.
  - Rachitis.
  - Thyroid insufficiency.
  - Enlarged adenoids and tonsils.

**Symptomatology.**—Enuresis, in about 55 per cent. of the cases, occurs only at night. About 40 per cent. are both nocturnal and diurnal, and about 5 per cent. are diurnal only. Incontinence of urine may occur once or several times during the night, or in milder cases days or weeks may intervene. Nocturnal incontinence occurs most commonly soon after the child goes to bed; at this time sleep is most profound. Enuresis, not being due to paralysis, or lack of development of sphincter muscles, does not have as one of its symptoms dribbling of the urine; on the other hand, the contraction of the bladder empties this organ as thoroughly as under normal conditions.

The urine should be examined with reference to increased acidity and the presence of bacteria, crystals, and other causes of bladder irritation; infection with the colon bacillus is a not uncommon cause.

**Prognosis.**—The prognosis as to ultimate recovery in nearly all cases is good. The great majority can be cured by careful systematic treatment within a period of two to six months. Even untreated cases, as a rule, recover by the seventh year of life. A small percentage resist all methods of treatment, and may persist even into adult life.

**Treatment.**—General Treatment.—In the treatment of no other neurosis of childhood is it of more importance to remove every possible cause of *reflex irritation.* It is a waste of time to begin medical or other treatment until a most careful search for reflex factors has been made. Phimosis should be relieved by circumcision, or by stretching the prepuce and carefully uncovering the glans; an adherent or contracted prepuce must not be allowed to persist. Genital, vesicle and rectal irritations, from the causes previously named, should be removed by appropriate medical or surgical treatment, and sources of reflex irritation in the throat, nose and eye should receive attention. Digestive disturbances of all kinds should be removed by proper medication.

The *diet* in all cases is important, even though the intestinal functions be normal. It is a good, general rule to exclude sweets, pastry, coffee, tea, beef juice, beef tea, and alcohol. The amount of nitrogenous food stuffs must be regulated to suit the individual case. Well-nourished children of gouty diathesis, having a tendency to acid urine and high specific gravity, should be given meat and eggs sparingly, but with children suffering from tuberculosis and other forms of chronic malnutrition these foods are indicated. In this latter class of cases tonics, such as cod-liver oil, iron and arsenic, may be of value, and fresh air, night and day, is important.

The child should be protected from *excitement* and *nervous strain;* should not be permitted to go to school; should be put to bed early and should have the whole routine of his daily life carefully regulated. He should neither be punished nor threatened with punishment for wetting the bed. He should, however, be made to understand the importance of overcoming this habit by retaining his urine for as long a time as possible during the day. If the child can be taught to accustom the bladder to hold

considerable quantities of urine for some hours during the day, the habit on the part of the bladder of discharging urine when only partly filled may not be carried over into the night.

In nocturnal incontinence of urine the child should take as little fluid as possible after four o'clock in the afternoon, and should be awakened to empty his bladder about an hour and a half after going to bed. The foot of the bed should be raised so that the child's shoulders will be lower than his hips, and he should, if possible, be prevented from sleeping on his back; incontinence of urine does not occur so readily when the child sleeps upon his side or stomach. A cold douche to the spine, once a day, may act as a tonic to the irritable spinal cord and assist in the cure of certain troublesome cases of enuresis; it may not, however, be well borne in nervous, malnourished children.

Medical Treatment.—Belladonna is the one drug which all writers recommend, and it is, without doubt, of great value. It should be remembered that belladonna may be given in comparatively large doses to children, and that to get results the dose must be gradually increased until the enuresis is controlled, or until pronounced physiological symptoms, such as dilatation of the pupils, dryness of the throat, or redness of the skin, are produced. In this event the drug is to be discontinued and subsequently administered in smaller doses. For a child of six years one may begin with a dose of three minims of the tincture three times a day; after three or four days the quantity may be increased one drop a day, until physiological symptoms are produced, or until the child is taking twenty-five or thirty drops in twenty-four hours. Holt says: "A convenient method of administration is to use a solution of atropin, 1 grain to ʒii of water, of which one drop (1/1,000 of a grain) may be given for each year of the child's age. For nocturnal incontinence this dose should at first be given at four and ten p. m.; after a few days at four, seven and ten p. m. Usually this may be gradually increased until double the quantity is given. A child of five years would then be taking ten drops (1/100 of a grain) at each of the hours mentioned. I have rarely found it advisable to go above these doses."

If the symptoms are benefited or controlled by the belladonna treatment this drug should be continued in smaller doses (one-half the size of the maximum dose) for months, or until the incontinence of urine has been controlled for a period of two or three weeks, and thereafter one dose should be given at bedtime for a period of four or five weeks. The belladonna treatment creates a tolerance on the part of the bladder which enables it to hold larger quantities of urine, and thereby materially assists in overcoming the habit of frequent urination.

Alkalies are indispensable in the treatment of cases in which there is a marked uric acid diathesis, and in which there is an excess of urates and acids in the urine. In these cases the belladonna should be given with benzoate of soda, or bicarbonate of potash or soda, and this prescription may be made more palatable by the addition of peppermint water com-

bined with some simple elixir. For a child of six years, five grains of either of these alkalies may be given after meals. It is better to prescribe the alkali, and the belladonna, or atropin, in separate bottles, so that the dose of the belladonna may be increased without increasing the alkali. In children having a periodic tendency to a return of acid urine these alkalies should be given over a long period of time.

Constipation, which is frequently present, may be overcome by a daily dose of phosphate, or sulphate of soda, given in carbonate waters to cover its taste.

In nervous, hysterical children, not of the acid urine type, bromid of potash given at bedtime may be combined with the belladonna treatment. It is, as a rule, not advisable to continue the bromid treatment for more than a week or ten days.

Strychnin has been very widely recommended in the treatment of troublesome cases of enuresis. It is perhaps of most value in those cases in which the incontinence occurs during the day as well as during the night. It should be combined with the belladonna treatment. It is perhaps contraindicated in nocturnal incontinence occurring in children having an exaggerated reflex irritability.

Urotropin in ½- to 1-grain doses three times a day is of decided value in those cases having a highly acid urine produced by a colon infection of the urinary tract.

Williams has noted the fact that a small group of cases respond readily to the judicious use of thyroid therapy. He recommends that undersized children having a subnormal temperature, a poor peripheral circulation and presenting a high arched palate, adenoids and enlarged tonsils and who do not respond to other methods of treatment should be given ½ grain of dried thyroid twice a day. In suitable cases a marked improvement occurs within a week and a cure rapidly follows. Williams further notes that with the disappearance of the enuresis the child rapidly gains in weight.

## CHAPTER LXXVII

### PSEUDOMASTURBATION IN INFANTS[1]

**Definition and Symptomatology.**—Pseudomasturbation is a syndrome in infancy and early childhood which has been described in medical literature under the titles, "Thigh Friction" and "Infantile Masturbation." It is commonly accomplished with the child lying on its back; the thighs are flexed, crossed and pressed tightly together, closely embracing the external genitalia; in this position the infant makes a wriggling, or up-and-down body movement, and rubs its thighs together. In other instances

[1] This chapter is taken, with slight modifications, from the author's paper on this subject in the *Archives of Pediatrics*, August, 1907.

the genitalia are rubbed with the hands or feet, or against some piece of furniture or other foreign object. These movements are apparently attended by a pleasurable excitement; the face is flushed and there is a marked increase in the general nervous tension. Following this act, which continues for a few minutes only, there is general relaxation, accompanied by mild perspiration, quiet contentment, and, in some instances, sleep. These attacks may occur many times in twenty-four hours, or days or weeks may intervene between them.

**Etiology.**—Age is the most important etiological factor. In the female the urinary bladder, the rectum and the external genitalia, including the clitoris, the labia majora and labia minora, are all derived from the same membrane, viz., the mesoderm of the allantois and cloaca. In the male analogous structures are derived from the same source.

The internal genital organs, including ovaries, uterus and vagina in the female, and analogous structures in the male, are derived from the Müllerian ducts and the genital ridges; and although these are of mesodermic origin, they are developed quite independently of that portion of the mesoderm which is being transformed into the urinary bladder, the rectum, and the external genitalia. The Müllerian ducts and genital ridges make their appearance later than the allantois and are united with it.

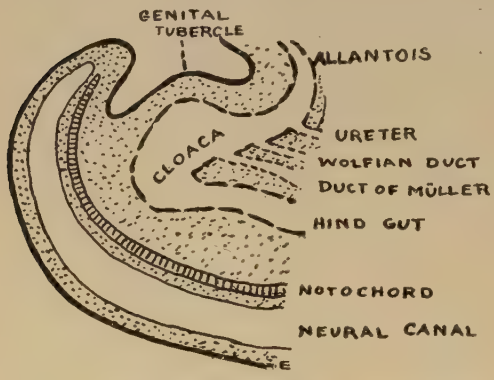



Fig. 87.—Embryo of 10 mm. (About Five Weeks).

Key: —— Ectodermic Tissue; - - - - Entodermic Tissue; . . . . Mesodermic Tubes; Shading = Mesodermic Tissue. (H. L. Woodward.)

The accompanying drawings illustrate the common origin of the urinary bladder, the rectum and the external genitalia, and also show the entirely different origin of the internal genital organs. The union between these groups of organs takes place about the fifth week of embryonic life, but there is a marked difference throughout embryonic life in their anatomical and physiological development.

The bladder, rectum, and external genitalia are rapidly developed, so that, at birth, the rectum and bladder have reached a fair state of physiological competency; and the external genitalia, being developed from the same structures, have been carried along in their evolution until they also have reached a considerable degree of development; the clitoris itself is almost as large and as sensitive as it becomes later in life. This, however, is not true of the internal organs of generation, which at birth are in a very incomplete state of anatomical and physiological development; and the rudimentary condition of these organs, according to Otto Küstner,[1]

[1] "Lehrbuch der Gynäkologie," 1904.

continues in the girl until she is ten years of age.[1] He says: "From birth until the beginning of puberty there is no real change in the genital tract of the girl. The uterus and vagina during this period undergo no development."

The close anatomical and physiological relationship existing between the bladder, rectum and external genitalia of the infant is still further shown in the nerve supply of these organs, which is practically derived from the same source, viz., the third, fourth and fifth sacral nerves and the mesenteric, sacral, and hypogastric plexuses of the sympathetic. These facts explain why the external genital organs of the infant, a few months after birth, are capable of responding to reflex excitation originating in any of the above-named parts, and why this excitation finds expression in producing a miniature syndrome so like true masturbation that one must conclude that this portion of the infantile genital system, which is later in life to come in closer touch with the fully developed internal sexual organs, must even at this early date in its development have impressed upon it the peculiar physiological function, which makes it respond to reflex excitation by mimicking the sexual orgasm.

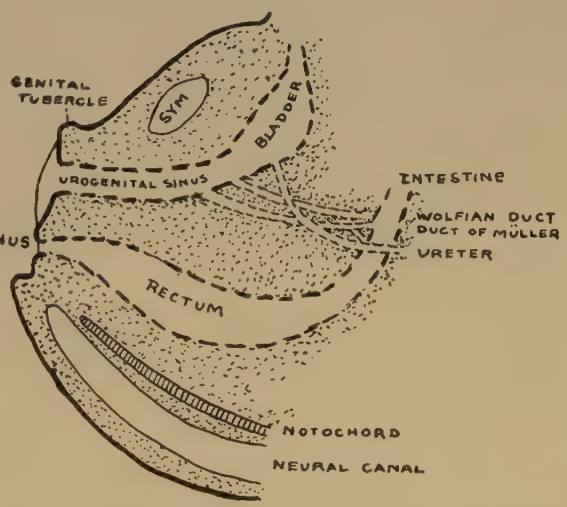



FIG. 88.—EMBRYO OF 25 MM. (ABOUT NINE WEEKS.)

Physiological functions go through various stages of evolution in the embryo, so that at birth most of them are developed to a state of physiological competency; this is not true, however, of the functions of the internal sexual and reproductive organs, which, as previously stated, are at this time both anatomically and physiologically in a very low state of development; so low, in fact, that they are as yet not endowed with physiological functions. In the infant, therefore, while we may have produced by reflex excitation of the external genitalia a syndrome which mimics the syndrome of true masturbation, we cannot have the fully developed orgasm, or a syndrome that equals true masturbation in the profundity of its sensations, or in the injurious effects it produces on the general nervous system.

In the child, after ten years of age, the internal sexual organs undergo rapid anatomical and physiological development, and during these years of development the intense feelings which accompany the sexual act may

be evoked by reflex excitation. This is the beginning of true masturbation. I do not mean to say, however, that true masturbation may not occur in certain children before they are ten years of age. Heredity and long-continued reflex excitation may cause a premature development of the internal sexual organs, carrying with this development a sexual precocity, which may make true masturbation a possibility in some children at a much earlier age. Pseudomasturbation, however, occurs as early as the fourth month, and the average age of onset of this neurosis, in my cases, is sixteen months.

Sex.—The majority of cases of true masturbation occur in male children, while of 60 cases of pseudomasturbation 55 occurred in female and 5 in male infants.

Habit.—The habit which is formed by the practice of pseudomasturbation becomes after a time one of its most potent etiological factors. No such sensations can be produced by exciting any other nervous mechanism in the body. In the first instance the excitation may be purely accidental, or it may have been caused by some local irritation; but after a time the frequent excitation of this nervous mechanism makes it more irritable and more easily excited, so that very slight reflex excitation is capable of producing a paroxysm of pseudomasturbation.

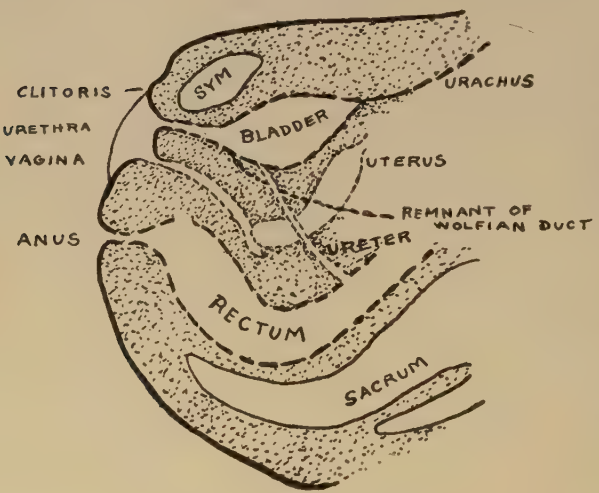



Fig. 89.—Child at Birth.

In the older child, *environment* may act as an etiological factor by throwing children together, offering the opportunity for imitation. It may also act by surrounding the child with an atmosphere of immorality and vice, which offers no restraining influence upon the development of this habit. In the infant, environment may predispose by producing bad hygienic surroundings, which may mean uncleanliness and lack of care of the genital organs, with increased local excitation, or it may mean malnutrition and other causes of general nervous irritability. The fact should also be noted that unscrupulous nurses sometimes teach infants the habit of pseudomasturbation as a means of quieting their fretfulness.

In fully three-fourths of the cases there is a distinct *neurotic inheritance;* in infants suffering from hereditary neuroses the reflex causes of pseudomasturbation may be very slight; so slight, in fact, as to escape

observation. A gouty inheritance may also predispose to this condition by producing in infants a tendency to periodic attacks of acid urine. I have had under observation a number of such cases where there was a return of the pseudomasturbation with every return of the attacks of acid urine, from which these infants suffered.

Malnutrition in infants suffering from pseudomasturbation influences very much the severity and frequency of the attacks. An attack of enteritis, influenza, or any other acute disease, which causes a rapid deterioration in general health, will produce a return of the habit, which can again be relieved only by complete convalescence from the intercurrent disease.

Direct Causes.—Irritation of the nervous mechanism which controls the sexual organs is the all-important exciting factor in the development of pseudomasturbation in infancy. The site of this irritation in the vast majority of cases is in the genitourinary organs, the rectum or the lower portion of the large intestine.

**Prognosis.**—This is good. I am convinced that pseudomasturbation occurring in infants under two years of age will almost always get well under proper treatment. The tendency in this disease is to spontaneous recovery, and the average length of time required to bring about this result is about nineteen months. The disease is a habit neurosis, and time, with a normal development of the nervous system which tends to stability and greater inhibitory control, is the most important factor in the cure of the worst cases. There is almost no connection between pseudomasturbation in infancy and true masturbation in later life. It is possible, however, that a badly neglected case of pseudomasturbation occurring in a strongly neurotic infant may continue until it becomes one of true masturbation in the child.

There is, I believe, no relationship between pseudomasturbation and epilepsy. The two conditions may coexist, and one can understand that the neurotic conditions which produce or underlie epilepsy may predispose to pseudomasturbation, but surely pseudomasturbation as here differentiated from true masturbation cannot be classed among the causes of epilepsy.

**Treatment.**—In the treatment of pseudomasturbation, as in the treatment of all habit neuroses, it is imperative that the habit be INTERRUPTED as soon as possible. The importance of this cannot be overestimated. The habit, whatever may have been its original exciting causes, has been engrafted upon the nervous system, and an interruption breaks into and helps destroy the habit, and in this way makes for the permanent cure of the affection. The accomplishment of this purpose, in some cases, is a matter of great difficulty. In the vast majority of cases, however, it is a comparatively easy matter.

As this act is performed, as a rule, while the infant is lying down, and commonly when it awakens from sleep, and when the parts are more or less irritated by the soiled diaper, it is imperative that a careful nurse, by constant watching, shall be present to forcibly prevent the act by tak-

ing the child up, changing the diaper, cleansing the parts, and dusting them with a soothing powder. The watchfulness of the nurse should continue throughout the waking hours of the child, so as to keep the parts always clean, dry, and free from irritating discharges. The child should be kept in a sitting posture as much of the time as possible, and even when taken for an outing should, if old enough, be carried about in a go-cart in preference to the ordinary baby carriage; the object of this is to keep the child in the position which least tempts it to practice the act, and the nurse should be directed to forcibly interfere at all times to prevent its accomplishment.

In children over two years of age mild punishment is sometimes very effective, and the child, when old enough, should be given to understand that it will be rewarded if it abstains from the habit. Moral suasion should be practiced with older children. It is evident that the above treatment can only successfully be carried out by an ever-watchful, patient, judicious nurse.

In the more severe cases forcible restraint during sleep may be necessary, as the infant cannot be watched constantly during the long hours of the night, and it may on waking practice this habit. Forcible restraint may be practiced in many ways. No special device is suitable to all cases. But if the physician is sufficiently impressed with the necessity for this method of treatment, the particular mechanical device by which the end is to be accomplished may be left to his ingenuity. If the infant sleeps in pajamas the heels of this garment may be fastened by safety pins to the mattress in such a manner as to hold the legs apart, and prevent the flexion of the thighs; at the same time the child's body is prevented from slipping down in the bed by a ribbon stretched from the back of the pajamas to the head of the bed. In younger children a large diaper may be folded, as suggested by Kerley, so as to prevent the thighs being approximated. Many writers have recommended heavy mechanical devices resembling fracture frames, into which the child is tied when it is put to bed. The profound sleep of the young child lends itself to this mode of treatment, and the patient quickly becomes accustomed even to such cumbersome appliances as double thigh splints with a separating footboard. It must indeed, however, be a very severe case to justify this form of apparatus.

When one has settled upon a plan for interrupting the habit, he should next turn his attention to the removal of all local reflex causes of irritation. In the male infant, phimosis and preputial adhesions should be treated, and in the female infant the preputial hood should be separated from the clitoris; vulvovaginitis and all irritations of the vaginal orifice should be treated. Pinworms, diseases of the rectum, local eczema, and, in fact, all abnormalities of the rectum and genitourinary organs should be removed, and the child's clothing should be carefully arranged so as not to produce local irritation.

Too much stress cannot be laid upon the importance of removing all

possible sources of local irritation of the nervous mechanism which controls the genital organs, as the reflex factor is not uncommonly the most important, not only in starting, but continuing, the habit of pseudomasturbation.

I wish, however, to call special attention to increased acidity of the urine as a potent reflex factor in many of these cases; I believe it is the most important of all reflex factors; it was present in one-third of my cases. This condition may be treated by benzoate of soda and tincture of belladonna put up in some palatable non-irritating vehicle. The alkali and the belladonna, the latter in small doses, should be given over a long period of time when there is any tendency to continuous or periodic acidity of the urine.

General Treatment.—Many cases, especially those over two years of age, are benefited by bromid of potash and belladonna given at bedtime. This treatment is especially applicable in those cases where the habit is practiced during the night.

An atmosphere of quiet and rest must, if possible, at all times surround the child. The importance of this injunction is as great in this as in the treatment of any other neurosis.

By the treatment above outlined it is possible in practically every case to control the habit, but it must be remembered that this treatment must, with more or less rigor, depending upon the severity of the case, be kept up not only for months, but sometimes for two, three, or even four years. Where the treatment, however, is carefully looked after one may count upon a permanent cure in the great majority of cases within one or two years. In those that are less carefully looked after four or five years may be necessary to accomplish a cure. One must recognize, therefore, that when the above treatment has been put into operation, and the habit controlled, the patient has been placed under conditions where time, by strengthening the stability and inhibitory control of the nervous system, will accomplish a cure. It, therefore, becomes important at this stage of the treatment to guard carefully the child's general nutrition, treating any special form of malnutrition that may exist, and securing normal development by careful diet and proper hygienic measures, including an outdoor life. Cod-liver oil, iron, arsenic and other tonics may enter into the treatment. It is important that the child should be guarded against constipation and all gastrointestinal disturbances, as attacks of this kind almost always cause a recurrence of the habit in an apparently convalescent child. The daily bath, followed by a cold douche, has been used with success.

# SECTION XI

## *DISEASES OF THE NERVOUS SYSTEM*

### CHAPTER LXXVIII

### DISEASES OF THE BRAIN

### INFANTILE CEREBRAL PALSIES

Infantile cerebral palsies comprehend a group of palsies which, in their general clinical manifestations, are so similar that they are classed together. They are characterized by spastic paralysis and by various other disturbances of cerebral functions associated with a great variety of cerebral lesions. That symptoms so like in character can be produced by pathological lesions so unlike in character and affecting such different parts of the brain is due to the fact that these lesions, occurring so early in the life of the child, seriously interfere with the general functional development of the brain. These cases may be symptomatically grouped as *hemiplegia, paraplegia* and *diplegia.*

**Etiology.**—The lesions which produce spastic paraplegia and spastic diplegia almost always occur at or before birth, although the symptoms may not appear for months later, but they never make their appearance after the third year. The lesions which produce spastic hemiplegia may occur at or before birth, but they usually occur after birth. The symptoms of these postnatal palsies, as a rule, follow quickly the injury to the brain; in some instances, however, the palsies, if slight, may not be discovered for months or years later.

PRENATAL PALSIES are due to traumatism, such as may result from a blow or a fall, or to uremic convulsions or exhausting illness during pregnancy, and hereditary defects transmitted by neurotic or alcoholic parents. These exciting causes may produce cortical hemorrhage, thrombosis, porencephalia, agenesis corticalis, and degeneration of the fibers of the pyramidal and lateral tracts. The lesions produced are usually extensive and result in diplegia or paraplegia.

NATAL PALSIES are due to asphyxia, false position of the head *in utero,* and traumatism from protracted labor and the improper use of forceps. There is no doubt, however, that the skillful use of obstetrical forceps has saved many children from serious brain injury at birth; this is especially true in those cases in which there is a premature discharge of liquor amnii. The lesion is due usually to meningeal hemorrhage producing subsequent lesions of the cortical motor area; more rarely the hemorrhage is directly into the brain substance and is followed by lack of functional

development and by inflammatory and degenerative changes on the part of the brain. These cases occur much more commonly in the first-born, and the paralysis which follows is commonly diplegic and paraplegic, but it may be hemiplegic.

Postnatal palsies are due to head injuries from blows and falls, to violent and protracted general convulsions, whatever may be their cause, to severe paroxysms of whooping-cough with the cerebral congestion which they produce, and to hereditary syphilis, meningitis, measles, influenza, and other contagious diseases. The lesions produced are cerebral hemorrhage, usually cortical, rarely intracerebral, thrombosis, embolism and hydrocephalus.

**Pathology.**—The primary lesion is usually *meningeal hemorrhage,* which may occur over any portion of the cortex. In diplegia and paraplegia it is bilateral, in hemiplegia it is unilateral, and is commonly located over the upper lateral surface of the brain, involving the motor areas in front of the fissure of Rolando. More rarely the initial lesion is an intracranial hemorrhage, a thrombus, an embolus or a chronic meningitis, producing hydrocephalus. Whatever may be the original cause, a meningoencephalitis occurs at the point of injury, producing softening, fatty degeneration and atrophy of cortical brain substance. Secondary sclerosis and scar tissue are in time left to mark the site of the original injury, and secondary degenerations may occur in the posterior and lateral columns of the cord. Porencephalia is very commonly found, especially in the prenatal cases; in this condition a cyst replaces a large portion of the brain substance. As a result of these lesions the functional development of the brain is retarded and epilepsy and imbecility may result.

The following table from Sachs, to whose careful studies we owe much of our knowledge of this disease, gives us an excellent classification of these cases from the standpoint of the age incident:

CLASSIFICATION OF INFANTILE CEREBRAL PALSIES (SACHS).

| Groups. | Morbid Lesion. |
|---|---|
| I. Paralyses of intrauterine onset. | Large cerebral defects. (Porencephaly.) Defective development of pyramidal tracts. Agenesis corticalis. (Highest nerve elements involved.) |
| II. Birth palsies. | Meningeal hemorrhage, rarely intracerebral hemorrhage. Later conditions: Meningoencephalitis chronica, sclerosis, and cysts; partial atrophies. |
| III. Acute (acquired) palsies. | Hemorrhage (meningeal, and rarely intracerebral); thrombosis (from syphilitic endarteritis and in marantic conditions); embolism. Later conditions: Atrophy, cysts, and sclerosis (diffuse and lobar).<br>Meningitis chronica.<br>Hydrocephalus (seldom the sole cause).<br>Primary encephalitis; polioencephalitis acuta (Strümpell). |

**Symptomatology.**—Hemiplegia.—This is the most frequent form of infantile cerebral palsy. It may be due to brain lesions occurring at birth or in early childhood, and the symptoms which announce the onset of the paralyses differ greatly in the natal and postnatal varieties.

When the injury to the brain occurs after birth, the most common period of incidence is from the sixth to the eighteenth month. The onset is almost always announced by severe general convulsions, which may be repeated at intervals over a number of days, and in the more severe cases an intervening coma occurs. High fever and vomiting usually accompany the initial convulsion, and they may persist throughout the convulsive period. It is now the generally accepted opinion that in some instances the acute brain lesions may be the direct cause of the convulsion, fever and vomiting. In other instances a severe convulsion from toxemia, whooping-cough or other causes may produce the cerebral hemorrhage, which in turn may directly aggravate the convulsion and other symptoms. In either event the onset is the same, and these symptoms are quickly followed by the characteristic paralysis. The more violent the onset the more marked will be the subsequent paralysis.

Fig. 90.—Hemiplegia From Cerebral Hemorrhage. (Sachs.)

When the injury to the brain occurs at birth this fact may be announced by cyanosis and convulsions during the first days of life. Following this acute cortical irritation the nervous symptoms may subside and the subsequent symptoms of the brain injury may await the development of the pyramidal tracts and the functional development of cortical and other brain centers. With the development of the myelin sheaths of the fibers of the pyramidal tracts during the first few months of life, the brain of the infant is put in closer communication with the spinal cord, and, as a result, there may slowly develop after the third or fourth month a spastic hemiplegia, or a more extensive paralysis, and late convulsive disorders may also occur. In some of these cases the primary injury to the brain may escape notice, and later an insidious spastic paralysis may develop, and the subsequent history may be very like those postnatal cases which are ushered in with violent convulsions, to be followed at once by a well-marked paralysis, except that in these latter cases the symptoms due to agenesis of the higher nerve centers are not usually so pronounced. These early symptoms in both natal and postnatal cases,

whatever may be the character of their onset, are followed by a spastic hemiplegia, which may involve the face, arm and leg of one side of the body. It is usually more marked in the arm. The amount of incapacity in the paralyzed side will vary with the extent of the brain lesion. In mild cases it may come on after school age, and may be only a slight muscular weakness developed by exercise. In severe cases it may be so great as to render the arm and leg useless.

*Muscular contractures* are the characteristic symptoms that differentiate this from the flaccid paralyses; the joints are bent and held more or less rigid; the forearm is pronated and flexed on the adducted arm, the wrist is drawn downward and inward, the hand is clenched, the fingers strongly flexed toward the palm, the knee bent, the foot extended downward and rotated inward, and the toes contracted. More or less recovery takes place in the paralyzed parts, especially in the leg, and as the patient regains the power of walking he has a spastic gait, dragging his toes and swinging his leg. In some instances fairly good control of the leg is finally obtained, leaving only a slight muscular weakness. But the contractures of the arm are more permanent. In a large percentage of the cases there develop in the paralyzed limb rhythmic tremor, choreiform movements, athetosis, or associated movements. In the latter condition the paralyzed arm imitates the movements of the good one.

All of the reflexes in the paralyzed extremities are greatly exaggerated, the kneejerk being especially valuable as a diagnostic sign, a slight tap upon the tendon producing a maximum contraction. The Babinski and allied signs are usually present. There is more or less lack of development in the paralyzed parts as time goes on, which results in shortening and shrinking of the limb without muscular atrophy. All of the paralyzed muscles respond in a normal manner to electrical excitation. Motor aphasia is usually associated with right hemiplegia, but if the lesion occurs before the child has commenced to talk, then speech is late in development. As Sachs has noted, however, aphasia in the young child may also be associated with left-sided hemiplegia. In course of time the child, as a rule, slowly regains or develops the faculty of speech.

*Epilepsy* occurs in from 30 to 50 per cent. of these cases. It may begin within a few weeks after the onset of the paralysis, or it may not appear for years. Sachs' valuable studies have thrown much light on the relation of epilepsy to early spastic palsies which have disappeared, or which were perhaps so slight at the time as to almost escape unnoticed, and his advice to carefully investigate every case of epilepsy with reference to its possible origin in an early cerebral hemorrhage will ofttimes reveal the cause of what would otherwise be considered as cases of idiopathic epilepsy of obscure origin. Exaggerated reflexes and weak muscular action on one side of the body, when associated with epilepsy, are strongly suggestive of an early cerebral lesion. The epilepsy may be of either the grand mal or petit mal type.

*Feeblemindedness* is one of the most frequent and distressing symp-

toms of this disease. Complete imbecility or slight mental weakness may result, and between these two extremes we may have every grade of mental defect. The most complete imbecility is usually found in the cases of diplegia and paraplegia. The hemiplegic cases may apparently retain their normal mental power, but, as a rule, they are not able to keep pace mentally with normal children in the severe strain that comes with advanced school work. The mental improvement in these cases should occur early, if it is to occur at all, and it is futile to hope for further intellectual development in children eight or nine years of age who have been in a state of comparative imbecility for years. Deaf mutism, blindness and hemianopsia may occur.

Fig. 91.—Spastic Diplegia from Cerebral Hemorrhage. (Sachs.)

Diplegia.—This is a double hemiplegia, both arms and both legs being affected. It is perhaps the most common form of cerebral paralysis during the first six months of life. It is produced by natal or prenatal injuries, and is an extensive double brain lesion, which may be marked by convulsive seizures during the first days of life. The paralysis, however, may not occur for weeks or months later, but never develops after the third year. In this condition the mental defects are much more pronounced than in hemiplegia. Imbecility is the rule, and with this hopeless lack of mental development there is a marked lack of physical development. Many of these cases never gain sufficient control of their legs to walk. Others learn to walk with a spastic crosslegged gait between the sixth and the ninth year of life. Epilepsy develops in perhaps 50 per cent. of these cases. Except, however, for the extent of the paralysis and the increased severity of all the symptoms, this form of the disease runs a course similar to spastic hemiplegia just described.

Paraplegia.—Paraplegia is also a double spastic paralysis involving both legs, produced by a double natal or prenatal injury to the brain involving both leg centers. The lesion is, therefore, more circumscribed than in diplegia. Except for the fact that the paralysis is confined to the legs, its clinical history is very like that of cerebral diplegia.

Monoplegia.—Monoplegia is very rare. Most of the cases which at first glance present this form of paralysis are old cases of hemiplegia, in

which the leg has apparently recovered, leaving the arm contractured and paralyzed. A closer examination of these cases will often show a weakness of the muscles of the leg and exaggerated reflexes, which are indications of the earlier paralysis of that part.

**Prognosis.**—In diplegia and paraplegia the prognosis is invariably bad. Fortunately many of the most severe cases die in infancy; the others remain more or less hopeless invalids, incapable of mental or physical development. The prognosis in hemiplegia, while not good, is much better, especially in those cases produced by lesions occurring after birth. Many of these postnatal hemiplegias recover with little or no mental defect, but more or less spastic paralysis of the forearm and hand remains in a great majority of the cases; but there is always a possibility that epilepsy may develop between the sixth and fifteenth year. The prognosis in hemiplegic cases dating from birth is not so good; in these the residual paralysis and the mental deficiency are more marked.

**Diagnosis.**—The differential diagnosis of cerebral palsies from the other paralyses of childhood has been considered under Infantile Paralysis.

**Treatment.**—In diplegia and paraplegia the underlying pathological lesions cannot be influenced by medical or surgical measures. It therefore becomes the duty of the physician to prolong the lives, modify the sufferings and control the nervous symptoms of these unfortunate children, many of whom live for years in a hopeless state of imbecility with fond mothers giving up their whole lives to care and nursing. The dietetic treatment is most important, since a slight constipation, intestinal fermentation, or a mild degree of intestinal toxemia may greatly aggravate the nervous symptoms, producing intense irritability, sleeplessness and even convulsions. The comfort of these patients depends largely upon the ability of the physician to keep the gastrointestinal canal in normal condition, and yet so feed them that they will be properly nourished. The sedative treatment is also important; sleeplessness, nervous irritability, muscular twitchings and convulsive disorders may require the use of bromids over a long period of time.

The hemiplegic cases offer a much more hopeful field for treatment. The convulsions and fever which mark their onset are to be treated with baths, ice-caps to the head, and the rectal or oral administration of chloral and bromids as directed under Convulsions. The subsequent treatment of these cases has in view the improvement of the general health of the child, the development of the paralyzed part, and the prevention of contractures. Good food, outdoor life, systematic massage and passive exercise to overcome contractures are the most important agents we have in accomplishing these ends; the massage should be general and should be given every other day for months or even years if necessary. Passive movements should be resorted to three or four times every day; these should be gentle and should be directed to overcoming the contractures; the contractured hand, forearm and leg should be gently extended five or six times at each sitting. The orthopedic treatment is also most important and should be

directed by an orthopedic surgeon. Properly applied braces, or the lengthening of and transplantation of contractured tendons, may put the child upon his feet, or give him better use of his arm, thus enabling him to expedite his recovery by active exercise. It is most important that children who are apparently approaching the normal in mental and physical development should not be pushed either mentally or physically. These apparently convalescent children should be carefully guarded over a number of years until it is plain that mental training will not injure them. Many of these children under the strain of school work become very neurotic, and, in some instances, they develop epilepsy.

## BRAIN TUMORS

The nature and position of brain tumors in children are shown in the following tables from Starr:

| Nature of Tumor. | |
|---|---|
| Tuberculous tumors | 152 |
| Glioma | 37 |
| Sarcoma | 34 |
| Gliosarcoma | 5 |
| Cystic | 30 |
| Gummata | 2 |
| Other varieties | 30 |

| Position. | |
|---|---|
| Cerebellum | 96 |
| Pons varolii | 38 |
| Centrum ovale | 35 |
| Basal ganglia and lateral ventricles | 27 |
| Cerebral cortex | 21 |
| Corpora quadrigemina and crura cerebri | 21 |
| Base | 8 |
| Fourth ventricle | 5 |
| Medulla | 6 |
| Multiple tumors | 43 |

**Symptomatology.**—The onset is insidious. The GENERAL SYMPTOMS develop slowly, may for a time come and go, and then gradually become permanent.

Headache gradually develops, but in time it becomes very intense, recurring in agonizing paroxysms, and in the interval between these severe attacks the pain may be dull and boring in character. Headache is one of the most significant symptoms, as it occurs early, is present in nearly every case, and increases in intensity as the tumor enlarges. It is of especial value in children, since headaches of this character from other causes are extremely rare at this age. Vomiting usually accompanies the headache. It may or may not be associated with nausea, is projectile in character, recurs without apparent cause, and is not in any way associated with the taking of food. Vertigo is associated with the headache and vomiting. The dizziness may be slight or extreme. If the patient is on his feet he may stagger and fall to the floor. Vertigo may be brought on in these cases by changing the position of the head. It is more frequent and more pronounced when the tumor is located in the cerebellum or in the pons. General or localized convulsions of every grade of severity may occur. In some cases they are absent altogether. They are more common

and more violent when the motor areas of the cortex are involved, and their diagnostic value depends largely upon their association with the other symptoms above noted.

Optic Neuritis.—The occurrence of the above symptoms should suggest an examination of the eyes and, if brain tumor exists, a double optic neuritis will commonly be found. It occurs in 80 to 85 per cent. of advanced cases, and is slightly more common in cerebellar tumors. Optic neuritis is, therefore, when taken in connection with the above symptom group, the most distinctive sign we have of brain tumor. It may be associated with partial or complete loss of sight and hearing; this combination of symptoms should suggest cerebellar tumor. As the disease progresses the intellect suffers, the child becomes dull, and may have but feeble mental capacity. Convulsions, stupor, coma and unconsciousness occur before death.

Localizing Symptoms.—To the above symptom group are added the symptoms which result from a disturbance of brain functions, which vary with the location of the tumor. These localizing symptoms, however, are the same in the child as they are in the adult, and do not, therefore, demand consideration here.

**Diagnosis.**—Tumors should not be confused with abscess of the brain; the latter is an acute febrile process characterized by chills and fever and associated with some septic process, which can usually be located. It should also be remembered that brain tumors are commonly tuberculous, and other evidences of this disease usually precede the development of the brain tumors. An examination of the cerebrospinal fluid should always be made, as it may help to exclude the various forms of meningitis.

**Treatment.**—The only cases that are at all influenced by medical treatment are those due to syphilis, and, although these are rare, it is perhaps advisable to give every case the benefit of antisyphilitic treatment. If the symptoms are not improved by a course of mercury and iodid of potash, then the only hope lies in surgical intervention. In most cases it is necessary to trephine and make an exploratory investigation before the advisability of a radical surgical operation can be determined upon. A small percentage of these cases are improved or cured by the removal of the tumor.

## ABSCESS OF THE BRAIN

Abscess of the brain is a rare disease. In childhood it is usually secondary to chronic otitis media or mastoiditis, but may also result from fractures of the skull, septic processes in the frontal and ethmoid sinuses, and more rarely from septic foci in remote parts of the body. Abscesses are most commonly located in the temporosphenoidal lobes, the frontal lobes and the cerebellum. As a rule only one abscess exists, and this may be so small as to almost escape observation, or so extensive as to destroy the greater portion of a lobe of the brain. In rare instances the abscess becomes encapsulated and the symptoms gradually disappear.

**Symptomatology.**—Brain abscess commonly begins with severe pain in the head, paroxysmal in character, associated with projectile vomiting, chills and fever. Irregular septic fever, when associated with recurring chilly sensations, pain in the head and vomiting, is a very important symptom. If the above symptoms occur in a child suffering from disease of the internal ear, from mastoiditis, or septic infection of the frontal or ethmoidal sinuses, there is every reason to suspect that an abscess of the brain is developing and, if an examination of the eyes reveals an optic neuritis, that suspicion will, in a large degree, be confirmed. Localizing symptoms may also occur, such as aphasia and paralysis, or disturbances of function of the cranial nerves.

**Diagnosis.**—In many instances it is impossible to make a diagnosis, but mistakes will be many times avoided by keeping in mind the acuteness of the above symptom group and remembering especially that the headache and vomiting are associated with symptoms of general sepsis, such as chills, irregular fever, and a well-marked leukocytosis, and that the cause of this sepsis can commonly be located in the ear, mastoid or sinuses of the face. A careful bacteriological examination of the cerebrospinal fluid should be made to exclude the various forms of meningitis.

**Course and Duration.**—While the onset of abscess of the brain is not always sudden, after the disease is once fully developed its course is, as a rule, rapid, terminating, in the vast majority of instances, fatally within two or three weeks. The later stages of this disease are marked by subnormal temperature, stupor, coma, slow pulse and marked disturbance of the respiratory rhythm.

**Treatment.**—The treatment is purely surgical. All cases due to trauma, otitis media, mastoiditis and sinus affections should be operated upon at the very earliest time possible. Cases that cannot be relieved by surgical measures are to be treated in a purely symptomatic way, and opiates should be given, if necessary, to relieve pain.

## CHRONIC INTERNAL HYDROCEPHALUS

Chronic internal hydrocephalus is due to increase of serous fluid within the ventricles of the brain, resulting in compression of brain substance against the bony walls of the cranium. Under this pressure the cranium enlarges, if bony union of the sutures of the skull has not taken place; this chronic internal form is commonly spoken of as *"hydrocephalus."* (Acute hydrocephalus occurs in association with meningitis, especially the tuberculous form, and its symptomatology is inseparably connected with the acute meningeal process of which it is a part, and even in those rare cases where the inflammation is confined to the ependyma or lining membrane of the ventricles, the clinical picture is that of meningitis, the symptomatology of which has been elsewhere considered.) In addition to the chronic internal form there is a condition known as chronic external hydrocephalus, in which the fluid accumulates between the dura and arachnoid

and compresses the brain against the floor of the cranial cavity; this is an inflammation of the dura and arachnoid commonly associated with defective development of the cerebrum. Its comparative rarity, however, and the fact that its symptomatology does not materially differ from the chronic internal variety, are sufficient reasons for disregarding its further consideration.

**Etiology.**—Chronic internal hydrocephalus may be congenital or acquired. Its etiological factors have not been fully determined. Inflam-

FIG. 92.—IDIOPATHIC HYDROCEPHALUS. An unusual degree of cranial enlargement. Circumference, 40 inches. (Willard Knowlton.)

matory lesions perhaps play an important rôle in producing the acquired form, and developmental defects are believed to be etiologically related to the congenital form. Czerny believes that pathological changes, which he found in the adrenal bodies, may be a cause of this condition. The exciting causes, whatever they may be, in some instances close the aqueduct of Sylvius and the openings between the ventricles of the brain, thus interfering with the circulation of the cerebrospinal fluid. Neurotic inheritance, congenital syphilis, and tuberculosis may be etiologically related to this disease.

**Pathology.**—The essential pathological condition is an accumulation of fluid in the ventricles, which may vary in quantity from a pint to four or five pints. The pressure of this fluid produces compression and atrophy of the brain substance. The distention is so great in severe cases that the brain is converted into cysts inclosed in thin walls of compressed brain tissue. Except in rare instances, where the sutures are firmly united, the skull is enlarged, and under this dilatation the sutures gap.

**Symptomatology.**—The most important symptom is the increase in size

of the head, which continues to grow larger as the disease progresses, until in a fully developed case the great increase in size of the skull is in marked contrast to the small, thin, wrinkled face. The forehead is high and pushes forward, the temporal and parietal bones spread outward, greatly increasing the lateral diameters of the head. The anterior fontanel is widely open, tense and pulsating, and the sutures leading from the fontanels may have opened under the pressure. The veins of the head are congested and prominent. The eyes protrude, are turned downward and have a peculiar stare, the cornea is partially covered by the lower lid, and the white sclera shows above; late in the disease, nystagmus, strabismus, and even total blindness may occur. The mentality of the child suffers as the disease progresses, its expression becomes dull and stupid, and it loses interest in its surroundings. Its body becomes more and more wasted, its arms and legs assume a more or less characteristic position, due to rigidity and contraction of the tendons and muscles. The arms are flexed, the fingers and toes contracted; the reflexes are exaggerated, tremor, choreic movements, convulsive twitchings and general eclampsia may occur, and the patient toward the end may lie in a helpless paretic condition.

The most pronounced and severe cases are, as a rule, of the hereditary form; in some of these death may occur *in utero,* or the head may be so large at the time of birth that the child dies during labor; in others the head is only slightly enlarged at birth, but slowly increases, until the characteristic symptoms are presented between the second and fourth months of life.

**Prognosis.**—Most of these cases die in early infancy; a few of the milder ones recover; of those in which spontaneous cure takes place the majority fail to reach full mental development.

**Treatment.**—In those cases in which hereditary syphilis is a factor, antisyphilitic treatment may favorably influence the progress of the disease, and since this is the only form of medical treatment that can be of any value, mercury by inunction and iodid of potash internally should be given a trial in every case where there is the slightest suspicion of syphilis. Lumbar puncture may in some cases give decided relief; if so, it should be repeated at intervals of two or three weeks. Aspiration of the ventricles followed by pressure bandages to the head may give temporary relief. Many other surgical measures have been tried, but all have been disappointing.

## MENINGOCELE, ENCEPHALOCELE, AND HYDRENCEPHALOCELE

These malformations represent different phases of congenital hernia of the contents of the skull. The openings through which the cranial contents protrude may be located in the occipital, the nasofrontal, the frontal, the temporal and parietal portions of the skull. The occipital region, however, is by far the most common location. In some instances a large portion of the occipital bone is absent, the defect extending upward along

the median line to the posterior fontanel or downward to the foramen magnum. Through this opening the contents of the skull protrude, forming a large or small tumor mass in the median line of the lower occipital region. When the hernia protrudes through an opening in the nasofrontal region the tumor occupies the bridge of the nose. The other most common sites for these hernias are along the lines of the cranial sutures.

### MENINGOCELE

Meningocele is the protrusion of the meninges or brain membranes through the opening in the skull. The protruding sac is commonly distended with fluid. This is the rarest variety of cerebral hernia. The opening in the bone is usually small and the tumor mass is small, pedunculated,

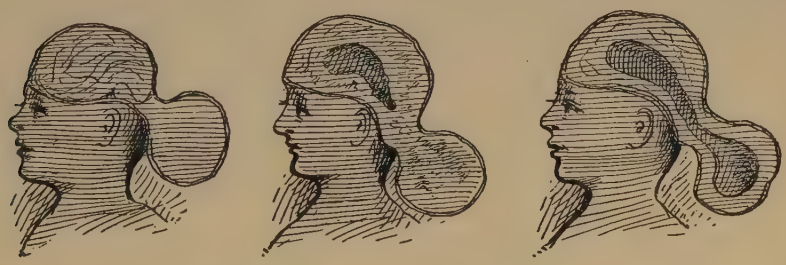


Fig. 93.—Meningocele, Encephalocele, Hydrencephalocele.

fluctuates on palpation, is translucent, presents no pulsation and is commonly reducible. This tumor mass, containing no brain tissue, is much more amenable to surgical treatment than other forms of cerebral hernia; the sac may be opened, its contents discharged and the opening in the skull closed. In all instances the radical surgical treatment of this condition is to be recommended, but it is advisable to delay the operation for some months, until the infant, on breast-milk, has commenced to develop physically and is in a fit condition to withstand the shock of the operation.

### ENCEPHALOCELE

Encephalocele is the protrusion of brain substance through the opening in the skull. The extruded cerebral substance carries before it the brain membranes. In this form of cerebral hernia the tumor mass is composed of brain substance not in communication with the ventricles of the brain; the only fluid that such a tumor may contain is on its outer surface between the brain tissue and the membranes covering it. In these cases the opening in the skull is large and the tumor comparatively small and not pedunculated. Pulsation in the tumor is very distinct; it is not translucent, and attempts at reduction are followed by symptoms of cerebral compression.

The treatment of encephalocele is very unsatisfactory; small tumor masses, especially in the frontal region, should be treated surgically; the

sac should be opened, the tumor removed and the opening in the skull closed. Larger masses occurring in the occipital region are less favorable for operation, but, notwithstanding the great mortality which follows the removal of these tumors, such radical surgical measures are followed by a greater percentage of recoveries than follow other methods of treatment. The injection of iodin, or other irritants, as well as the expectant plan of treatment, offers even slighter chances of recovery. The removal of the tumor is to be advised in all infants suffering from encephalocele who have lived past the sixth month of life, and during this time have gained in nutrition and have commenced to show symptoms of normal mental development. Large tumors associated with other congenital defects occurring in infants who fail to develop both physically and mentally should be considered inoperable.

### HYDRENCEPHALOCELE

Hydrencephalocele commonly occurs in the lower occipital region; this is the most frequent and the worst form of cerebral hernia. The tumor mass in these cases is made up of brain membranes, covering the protruding brain substance, which contains a cavity filled with fluid in direct communication with the ventricles of the brain.

As these cases are inoperable it is important that they should be differentiated from encephalocele, and this differentiation may be made with a fair degree of accuracy by the following symptom group: The tumor is large, sometimes five or six inches in diameter; it is faintly translucent; deep fluctuation is present; it is pedunculated, pendulous, irreducible, and its surface is irregular and offers little resistance to palpation. The skull is commonly deformed, giving to the eye the impression of imbecility. In doubtful cases deep aspiration of the tumor reveals the presence of fluid.

The prognosis is absolutely bad; surgical treatment is contraindicated, and symptomatic medical treatment can only promote the comfort and prolong the life of the patient.

## IDIOCY

Mental deficiency, imbecility and idiocy are terms used to represent various degrees of mental impairment due to congenital defects, disease and injury of the undeveloped brain of the infant, with an associated lack of development on the part of the general nervous system.

**Etiology.**—Idiocy may be either congenital or acquired, although it is difficult to conform to this or any other classification in describing a condition with such widely varying etiological factors.

Congenital idiocy is due to development defects of the brain, such as porencephalus, agenesis corticalis, and other little understood conditions. These cases represent the worst types of idiocy and are very frequently associated with other congenital malformations and with the stig-

mata of degeneration in other parts of the body. They are also etiologically related to parental alcoholism, syphilis, hysteria, insanity, epilepsy and chorea. It is believed that consanguinity of parents, as in the marriage of first cousins, may predispose to idiocy by exaggerating the family's neuropathic taint, thereby exaggerating congenital defects. Cerebral hemorrhage occurring before or at birth, and followed by spastic paraplegia and diplegia, is one of the common causes of mental deficiency and imbecility (see Cerebral Palsies). Microcephalus is associated with one of the worst types of idiocy and hydrocephalus may produce mental deficiency and imbecility.

The acquired forms of idiocy are due to the following causes: cerebral hemorrhage, meningitis, encephalitis, epilepsy, eclampsia, traumatism and asphyxia.

**Symptomatology.**—Mental deficiency, no matter how marked it may be, is rarely recognized during the first months of life, except in the Mongolian type of this disease. Defective infants are believed to be normal in the great majority of instances, until the time arrives when it is evident, even to the mother, that the infant does not handle its body, use its arms and legs, and otherwise act as normal infants do. When it is perhaps six months of age the attendants notice that its body is limp, and that it makes no effort to hold up its head or straighten its spine. As time passes it may become evident that the infant fails to distinguish between the faces that constantly surround it; it does not recognize its mother. During the second year of life, instead of uttering words that have been repeated to it, it makes strange sounds or perhaps utters shrill cries, and all of its actions are without purpose or intent. It fails to grasp at or take hold of its nursing bottle, is unable to lift itself in bed, and, even toward the end of the second year, makes no effort to walk. In the early stages of this condition these signs of lack of physical development are more evident than symptoms due to lack of mental development, but as the child grows older the mental defects become evident. The facial expression in almost every instance bears the mark of stupidity and lack of intelligence to everyone except the mother, and perhaps those who have been constantly associated with the child from birth. On the one hand the child may be stupidly amiable, never crying, easily amused, knowing no fear, making friends with everyone, and quite as happy when amused by strangers as when it is with its mother; or again it may be irritable, easily frightened and uncontrollable. The degree of mental deficiency will vary greatly with the extent and severity of the brain injury which produces it. In many of the acquired cases, especially those associated with cerebral hemiplegia, the child has a fair degree of intelligence, and the mental development in these cases may be greatly improved by careful, systematic training. In other instances, especially those associated with congenital brain defects, intellectual activity may be almost or totally lost. Such cases have no idea of personal cleanliness, eat their food in a ravenous manner when it is fed to them, fail to acquire the faculty of

speech, and make strange uncouth noises that have no relevancy to their surroundings.

**Prognosis.**—The prognosis in all of these cases is bad so far as total recovery is concerned, but from the standpoint of partial recovery the prognosis depends upon the character and extent of the lesion. The congenital cases due to defective development of the brain are hopeless. Those due to cerebral hemorrhage and associated with diplegia and paraplegia are also hopeless, but those associated with hemiplegia, as previously noted under Cerebral Palsies, may have a fair degree of development. Those due to inflammatory conditions of the brain and meninges may be only partially defective, and those cases associated with epilepsy may suffer from very slight or very marked mental deficiency.

**Treatment.**—Hopeless cases are better cared for in institutions, where they will be much happier than in their own homes, and where they will not have an unfavorable influence upon the other children in the family. Feebleminded or slightly defective children should be placed under the care and direction of teachers who have been especially educated for this kind of work. Under the care of intelligent specialists the best possible results in the mental development of these children can be obtained. The late Dr. Christopher, of Chicago, did excellent service by helping to establish a special system of education for defective children in connection with the public schools of Chicago. These defectives were separated from the normal children in the public schools, were properly classified and placed under competent instructors. Nearly all of the large cities of our country are beginning to recognize the importance of furnishing to the defective children of the poor an education which will develop the best that is in them and possibly make them self-supporting in after life.

### AMAUROTIC FAMILY IDIOCY

The etiology of this condition is unknown; it is congenital, and, although a rare disease, more than one case may occur in the same family.

At birth the child is apparently healthy, but at six or eight months of age physical and mental defects are observed. It does not use its body and limbs as normal children do, and shows absolutely no sign of mental development. Nystagmus occurs and blindness gradually results from atrophy of the optic nerve. An ophthalmoscopic examination shows red spots on a grayish-white opacity in the region of the fovea centralis. A condition of absolute idiocy is presented, the child has no mental perceptions, spastic paraplegia may develop, progressive emaciation occurs, and the disease invariably ends in death, usually before the end of the second year.

### MONGOLIAN IDIOCY

The cause of this condition is unknown, but its most striking symptom is the facies, which is characteristic of this disease, and by it the diagnosis is made. The facies consist in a Mongolian or Chinese type of

face, characterized by the downward slant of the palpebral fissures toward the nose, which is broad and low. The cheeks are full and high-colored; the skin and hair normal; the tongue, although not swollen, lolls out of the mouth as it does in the cretin. The head is flat, the fontanels remain open longer than usual, and the skull is brachycephalic and below the average in circumference. The hands are short; this is especially noticeable in the thumb and little finger; the latter curving inward over the ring finger is a sign of diagnostic importance in differentiating this disease from cretinism. The characteristic facies above described may be noticeable soon after birth, and by it the physician may be able to foresee the subsequent development of Mongolian idiocy.

These Mongols as they grow older show not only delayed physical development but marked lack of mental power. They teethe slowly and are late in getting control of their arms, legs and body, so that they may be four or five years of age before they walk with ease. From this time on their mental defects are much more noticeable, but, as a rule, they continue to slowly improve in intellectuality, being classed as very backward children. At three or four years of age they may understand what is said to them, be able to repeat simple words, play with their toys, be interested in their surroundings, and may finally reach a stage of mental development which enables them to look after their personal wants, observe ordinary habits of cleanliness, and even learn to read and write, but beyond this little is to be hoped for.

**Treatment.**—There is no medical treatment that favorably influences this condition. The treatment of these children, therefore, consists in looking carefully to their mental and physical development. Their mental development can be favorably influenced by placing them under the care and direction of competent teachers, who have been trained for this work.

### MICROCEPHALIC IDIOCY

In this form of idiocy the head presents a characteristic deformity. The circumference of the cranium is small, the forehead is very low and sharply recedes into a poorly developed occipital prominence, the fontanels are closed, the sutures prematurely ossified, and the face is proportionately large, giving the head a peculiar bird-shaped appearance, stamped with an expression of absolute idiocy. The primary pathological lesion in these cases is situated in the brain; a microcephalic brain may even be incased in a normal cranium. The small skull in most of these cases has absolutely nothing to do with arresting the growth of the brain. The arrested development of the brain, like the perverted development of the cranium, is due to developmental defects. In some instances there may be a lack of development of the whole brain. In other cases the occipital, the parietal or the frontal lobes may be undeveloped, and the small and prematurely ossified cranium is, as a rule, more commodious than the atrophied brain demands. Operation upon the skull, to increase the capacity

of the cranium and allow the brain to grow, is founded upon an erroneous conception of this disease, and does absolutely no good.

Microcephalic idiocy, as the term is commonly used, merely refers to a type of hopeless and almost complete idiocy in which the atrophied, diseased and deformed brain is inclosed in a microcephalic skull and does not definitely determine the character or location of the brain deformity. The multiplicity of brain lesions which exist in these cases explain the fact that in some instances the rest of the body may be normally developed and the patients may live past middle life, while in other instances spastic and flaccid paralysis may exist and the duration of the disease may be much shorter.

**Treatment.**—There is no treatment, either medical or surgical, that favorably influences the course of this disease.

## CHAPTER LXXIX

### MENINGITIS

Meningitis is an infectious disease producing inflammation of the pia mater and arachnoid membranes of the brain and spinal cord. For clinical reasons the various forms of this disease are here grouped under one heading.

In the present state of our knowledge it is impossible to make a satisfactory clinical classification of the different forms of meningitis. The syndromes presented by the various forms so closely resemble one another that a classification based on clinical phenomena alone is absolutely impossible. In this dilemma it is perhaps better for text-book purposes to adopt the classification now in vogue based upon clinical, bacteriological and pathological findings. It recognizes three varieties, the differential diagnosis of which can nearly always be made by an examination of the cerebrospinal fluid, studied in connection with the clinical syndromes which the various forms of meningitis present.

This classification is as follows:

1. Tuberculous meningitis, produced by the tubercle bacillus.
2. Epidemic cerebrospinal meningitis, produced by the meningococcus intracellularis.
3. Purulent meningitis, a term used to include all forms not produced by the tubercle bacillus or meningococcus.

### TUBERCULOUS MENINGITIS

**Pathology.**—This form of meningeal inflammation is produced by the tubercle bacillus and its general etiology is the same as that of other forms of tuberculosis. The meninges are soon studded with small gray tubercles usually attached to the blood vessels. A thick, yellow, inflammatory exu-

date forms over the base of the brain, and extends with the blood vessels in the sulci which lead toward the convexity of the brain. The ventricles are distended, producing internal hydrocephalus; the gradually increasing hydrocephalus produces intracranial pressure, pushing with considerable force the convolutions of the brain against the unyielding bony wall of the cranium. The brain tissue just beneath the meninges may contain caseous nodules which sometimes reach the size of a hen's egg. These tuberculous tumor masses may produce localizing symptoms. The meningeal inflammation may not only involve the cervical region of the cord, but may extend along the entire spinal canal.

Tuberculous meningitis occurs most frequently in infancy and early childhood; about 70 per cent. of the cases are seen between the end of the first and the fifth years of life. It is the cause of death in 20 to 30 per cent. of all cases dying of tuberculosis under five years of age, and at this period of life it comprises about 70 per cent. of all cases of meningitis, except when the epidemic form is prevalent. It is commonly secondary to lymph-node, pulmonary, and general miliary tuberculosis, but may result from tuberculous foci anywhere in the body, the blood and lymph streams being the carriers of the tubercle bacilli to the meninges; in nearly every instance the infecting organism is of the human type. It is rarely, if ever, a primary disease, but may spread from bony cavities in the face, nose and ear, with no preliminary tuberculous disease elsewhere in the body.

**Symptomatology.**—In infancy tuberculous meningitis is commonly a manifestation of a general tuberculosis and is not, as a rule, preceded by premonitory symptoms due to tuberculosis elsewhere in the body. After the second year of life, however, it is generally very insidious in its onset and is preceded by the symptoms of lymph-node, bone, lung or general miliary tuberculosis. Its symptoms, especially in children over three years of age, may be divided into three groups representing the stages of invasion, irritation and compression. It must be remembered, however, that while the symptoms, for clearness of presentation, are here described under these three groups, the symptomatology of this disease does not by any means always follow this regular course. On the other hand, the variability and irregularity of the symptoms are notable characteristics of tuberculous meningitis.

The STAGE OF INVASION is marked by fever, vomiting, constipation, great nervous irritability, sleeplessness, loss of weight and general prostration, none of which are especially characteristic of this disease; but a combination of some or all of these symptoms is especially suggestive when they occur in a child who has tuberculous foci elsewhere in the body, or who has lived under conditions which have repeatedly exposed it to the tuberculous contagion. The vomiting occurring and recurring without apparent cause is the symptom which gives special importance to this symptom group. The temperature during this period is usually overlooked, and varies from normal to 101°F. The Moro and von Pirquet skin re-

actions are nearly always present during this stage. These symptoms may continue, especially in older children, two or three weeks, and during this time the child may have days of apparent convalescence, but its general condition steadily grows worse until the stage of irritation presents the following more characteristic symptoms: An increase in the fever to 101° or 102°F. occurs, but the temperature, as a rule, does not run high unless there be other tuberculous lesions. The restlessness gives way to mental dullness and stupor, which may alternate with extreme nervous irritability and may be associated with a mild delirium; convulsions, either general or local, may occur; muscular rigidity, producing retraction of the neck and stiffness of the spine, appears; localized facial palsy and spastic paralysis of the extremities may come and go; Kernig's sign is present sooner or later in the majority of cases, but is not so frequent as in other forms of meningitis; Babinski's reflex is slightly more common in this form of meningitis than in any other, and is, therefore, in children over two years of age, a sign of some value in differential diagnosis. The pupils may be unequal and respond slowly or not at all to light; strabismus is a common and a very suggestive sign; nystagmus may be noted among the earliest eye symptoms, and the ophthalmoscope may reveal an optic neuritis with bright shining tubercles in the choroid. Vasomotor disturbances are common, red patches involving the ear or other portions of the body may come and go without apparent cause, and a red streak may be brought out by drawing the finger over any portion of the body; the petechial eruption is absent. The respiratory rhythm is disturbed; the respirations are irregular and marked by periods in which the respiratory movements are suspended; gradually a Cheyne-Stokes type of breathing may be developed. The pulse may be slow and intermittent. This stage may last a week, more or less, depending upon the age of the child and the severity of the disease. During this time there may be great variations in this symptom group. It is not an uncommon experience to find that a child that has been for a number of days profoundly stuporous and almost or quite unable to swallow food, rather suddenly recovers consciousness and again swallows his food in a normal manner. This apparent improvement causes the attendants to hope that the child is really better, when after a few hours the stupor returns and the whole symptom group gradually grows worse, until the third stage of the disease arrives with symptoms of cerebral compression. The coma deepens; unconsciousness is complete; the muscular spasm is relaxed; stiffness of the muscles of the spine disappears; general paralysis, permanent, and of the flaccid type, is widespread; deglutition becomes more difficult and at times impossible; the eyelids are partially closed, the pupils widely dilated, and the eyeballs turned upward. The pulse is very rapid and weak; the breathing becomes more rapid, more irregular, and the respiratory pauses are more noticeable. The temperature, which has run a low range throughout the disease, rarely rising above 102° or 103°F., toward the end of the disease may rise to 105° or 106°F. A deepening coma not infrequently accom-

panied by convulsions terminates the clinical picture. This stage lasts from a few days to one week.

The above symptom group is subject to many variations, largely determined by the age of the child and the virulency of the pathologic process. The average duration, not counting the vague prodromal symptoms, is two weeks in young infants and three weeks in older children; other things being equal, the younger the child the more rapid and the more violent will be the course of the disease.

**Diagnosis.**—The diagnosis of tuberculous and other forms of meningitis depends upon the careful study of the above symptom group in connection with the individual case; giving special attention to the family history,

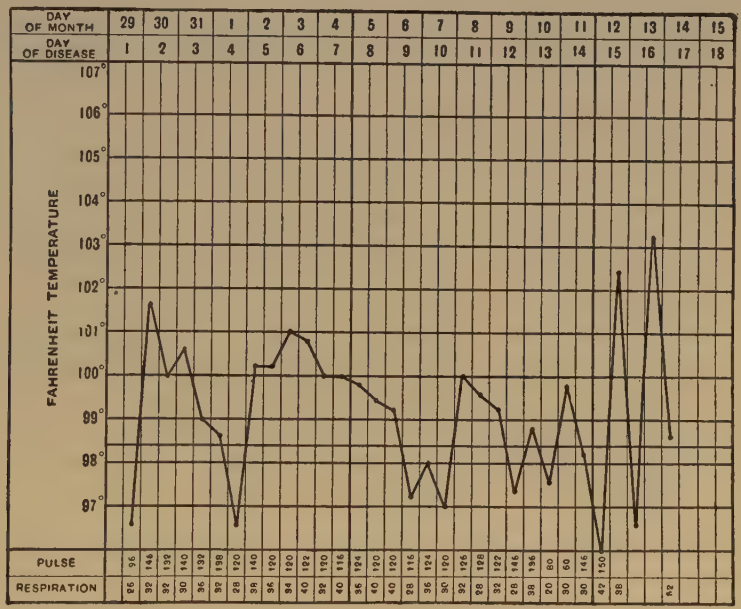



Fig. 94.—Tuberculous Meningitis in Child Twenty Months Old.

the opportunities for contracting the tuberculous contagion, the presence of tuberculous foci elsewhere in the body, the slow onset of the disease, the low range of temperature, the character of the breathing, the eye symptoms, the ophthalmoscopic findings, the presence of Moro's and von Pirquet's tuberculin skin reactions in the early stages, and, most important of all, *the finding of tubercle bacilli in the cerebrospinal fluid;* with careful technique this may be accomplished in the majority of cases. The diagnosis may still further be confirmed by producing tuberculosis in the guinea pig by inoculating it with the cerebrospinal fluid.

Concerning the diagnostic importance of a careful examination of the cerebrospinal fluid, Dunn states that: "The order in which the inferences are made during such an examination is as follows: if the fluid is cloudy, some form of meningitis is present; if clear, no form of meningitis can

be present, except the tuberculous. If the cell count is normal (under ten per cubic millimeter), no meningitis is present; if the cell count is increased (over ten per cubic millimeter and usually over one hundred per cubic millimeter), some form of meningitis is present. If, with meningitis present, the predominating cell is the mononuclear lymphocyte, the evidence points toward the tuberculous form; if the predominating cell is polymorphonuclear, the evidence points toward some other form. The differentiation of these other forms depends on the finding of the specific etiologic microorganisms in the cerebrospinal fluid. The diplococcus intracellularis and the influenza bacillus are recognized by their morphology and their Gram-negative staining reaction; the former also is frequently seen within the leukocytes. The pneumococcus, streptococcus, and staphylococcus are recognized by their morphology, and their Gram-positive staining reaction. The tubercle bacillus is recognized by its special staining reaction. The chief difficulty is in distinguishing the pneumococcus from the staphylococcus form, from the fact that the former frequently shows a chain formation, and its capsule is difficult to demonstrate in the cerebrospinal fluid. Nevertheless, this difficulty is not so great in the cerebrospinal fluid as under some other conditions. In meningitis the streptococcus usually forms long chains and its morphology is unmistakable, while the pneumococcus frequently is seen in short chains; its typical form as a lance-shaped diplococcus is usually plainly evident. The positive proof of the existence of tuberculous meningitis is also often difficult, as bacilli cannot always be found. We are obliged to depend on the increased cell count, with the preponderance of lymphocytes. Where the cell count is only moderately increased some doubt of the diagnosis will remain. Also the cell count in the cerebrospinal fluid is increased in some other conditions which, however, are not of a kind usually mistaken for tuberculous meningitis. The existence of an increased number of mononuclear cells is sufficient for ordinary clinical purposes, but if the bacilli are not found, it cannot be taken as absolute proof sufficient for scientific purposes."

**Prognosis.**—This is almost hopeless. A very few cases of recovery, however, with well-established diagnoses, are reported each year.

**Treatment.**—Since under existing forms of treatment the prognosis in these cases is almost hopeless, they should be treated symptomatically, as outlined under meningococcus meningitis. The specific serum, however, of this latter disease is of absolutely no value in the treatment of any other form of meningitis. The possibility, however, that there may be a mistake in diagnosis, and the fact that a small percentage of cases of tuberculous meningitis recover, should stimulate the physician to the application of all remedies which ameliorate symptoms and prolong life. Early and repeated lumbar puncture is believed by Dunn to exercise a favorable, and perhaps a curative, influence in rare instances.

## MENINGOCOCCUS MENINGITIS

Meningococcus meningitis is an acute infectious, feebly contagious disease caused by the diplococcus intracellularis meningitidis. It occurs both sporadically and epidemically, and is characterized by a general systemic intoxication and the symptoms of a violent inflammation of the pia mater and arachnoid membranes of the brain and spinal cord.

**Etiology.**—The SPECIFIC MICROORGANISM of this disease was first described by Weichselbaum in 1887. It is found in the mucous discharges from the nose and throat, in the blood and in the cerebrospinal fluid of infected individuals, and may also be demonstrated on the mucous membranes of the throat and nose of healthy individuals, who are closely associated with patients ill of this disease. These "meningococcus carriers," it is believed, may carry the infection to susceptible individuals. It is thought that this diplococcus finds its portal of entrance to the human body through the nose and throat, and is disseminated by the mucous discharges from the respiratory passages or by the careless handling of cerebrospinal fluid drawn for therapeutic or diagnostic purposes. But, notwithstanding the apparent danger of spreading the disease in this way, long experience has taught that there is little actual danger from contact contagion such as exists in the other acute infections. That the disease is but feebly contagious is demonstrated by the fact that these cases have always been treated in the general wards of our hospitals, and until the discovery of its specific cause, clinicians had scarcely suspected that there was any greater danger of direct contagion from this form of meningitis than from any other. The slight contagiousness may be due to the lack of individual susceptibility and to the "brief vitality" of the specific contagion. On the other hand, one must recognize the fact that the contagion is at times very widely disseminated, producing extensive epidemics. Minor epidemics have occurred in all of our large cities, and have also been reported from country districts and small towns remote from great centers of population. In the intervals between these epidemics the disease occurs sporadically throughout the land, now and then appearing in different parts of the same city, or perhaps as isolated cases in lone farm houses. The prankish vagaries of this contagion are yet to be explained.

AGE.—The disease is rare in infancy, although Rotch reports a case in an infant six days old. After the first year of life, however, it is not uncommon, and childhood is the period of greatest susceptibility. It is comparatively infrequent in the adult.

SEASON.—It occurs most commonly in the spring of the year; more cases are seen during March and April. It is less commonly observed during the summer months. It is believed that the low vitality of children at this period of the year may make them more susceptible.

**Pathology.**—This disease is characterized by an infection of the pia mater and arachnoid with the encapsulated diplococcus of this disease.

These microorganisms are found in the polynuclear leukocytes which are thrown out in great numbers, and in the cerebrospinal fluid which is greatly increased in quantity. These brain membranes are in the beginning intensely congested, and this hyperemia is followed by a serofibrinous and seropurulent exudate which collects at the base of the brain and in the ventricles and may extend over the cortex and down the spinal canal. The fluid found in the ventricles and cerebrospinal canal quickly becomes clouded with pus corpuscles, and later may become a distinctly purulent fluid.

In the foudroyant cases the patient may die before the inflammatory lesions are marked. In such cases the brain membranes may show only intense congestion with a serous exudate. In the less severe cases the process lasts longer and a decidedly purulent exudate may be present. In the more or less chronic cases lasting for weeks the pia mater and arachnoid are thickened and bound to the brain by an inflammatory exudate.

**Symptomatology.**—Onset.—In Huber's careful analysis of 100 cases, only 3 presented prodromal symptoms. In these the more acute symptoms were preceded, by a few hours, with malaise, headache, loss of appetite, indefinite pains, chilliness and slight rise of temperature. In all of the others the onset was sudden, and in many the exact hour when the acute symptoms began was named. There is perhaps no other disease in which the onset is so uniformly sudden and violent as it is in cerebrospinal meningitis. The disease commonly begins with vomiting, chilliness, severe headache, and rapid rise of temperature. To this symptom group is sometimes added convulsions, which are especially common in young children. A petechial eruption may occur. Spinal pain and tenderness associated with general hyperesthesia may cause the patient to cry with pain whenever he is touched. These symptoms are followed by tenderness and retraction of the postcervical and spinal muscles (opisthotonos), extreme nervous irritability and muscular tremor. Delirium quickly develops, and in severe cases this is followed by stupor, coma, convulsions and death. In the milder cases the same symptoms mark the onset, but the subsequent course of the disease is less severe. Fatal cases may begin with comparatively mild symptoms, and cases that recover may have a violent onset, but, on the whole, a severe initial general toxemia is commonly followed by a severe inflammation of the brain and cord. In older children the violence of the onset is more directly in proportion to the severity of the subsequent symptoms than it is in infants, to whom the open fontanels furnish a certain degree of protection from the early pressure symptoms of this disease.

Vomiting, which is commonly the initial symptom, is projectile in character and may persist for a number of days, but rarely continues throughout the attack. It is usually associated with constipation.

*Nervous Symptoms.*—The headache which is an early symptom is usually intense and is usually frontal, but may involve other portions of the head and may be associated with restlessness, irritability and photo-

phobia. The postcervical and spinal muscles become tense and tender and more and more retracted until a marked opisthotonos is produced. Irritation of the sensory roots of the spinal nerves produces tenderness on either side of the spine and a more or less general hyperesthesia of the skin, which causes the patient to cry out with pain when touched. The irritation of the motor nerves, which produces retraction of the head and backward curvature of the spine, may also produce a tonic contraction of the muscles of the arms or legs, resulting in the drawing up, under more or less tension, of these extremities. The reflexes associated with meningeal irritation are well marked. Kernig's sign is rarely absent. The Babinski reflex, which is of little value in children under two years of age, can usually be demonstrated, and the tâche cérébrale is present.

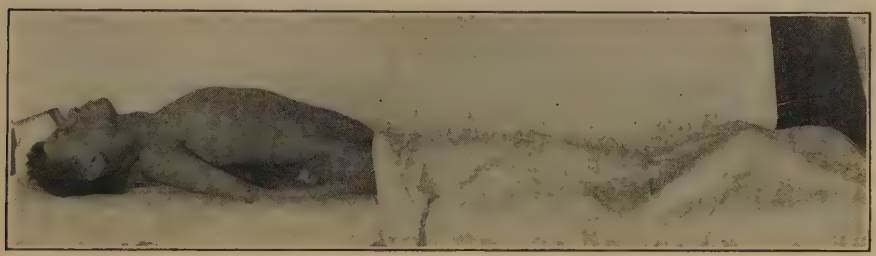


FIG. 95.—OPISTHOTONOS IN CEREBROSPINAL MENINGITIS.

These reflexes, while of importance in confirming the diagnosis of meningitis, are of little value in differentiating this from other forms of meningeal inflammation. Delirium, following chilly sensations, is an early and common symptom; in the milder cases it may quickly subside, and the patient may remain conscious throughout the course of the disease. In the more severe cases the delirium may become so violent that restraint is necessary, and stupor and coma may supervene. The patient may come out of the coma to again become actively delirious, and this delirium may gradually subside as convalescence is established. A prolonged comatose condition not infrequently occurs in fatal cases.

*Fever.*—With the onset the fever rises suddenly. Within the first twenty-four hours it may reach 104° or 105°F. This sudden rise in the temperature, accompanying the initial symptoms of vomiting and headache, is of diagnostic importance, but thereafter the temperature curve is of little diagnostic or prognostic importance, as it is subject to such variations. After the initial rise, the temperature may fall almost to normal within a few days. Again it may rise to 104° or 105°F., and its subsequent irregularities, which may include subnormal temperatures, followed by high fever, give little information as to the progress of the disease. In prolonged cases the temperature may continue for many weeks and even months.

The *pulse* in children is rapid, commonly ranging between 120 and 160. *Respiration* is accelerated; as the disease advances it may become irregular, and Cheyne-Stokes breathing rarely occurs.

A *petechial eruption* may occur as dark-red points widely disseminated over the skin of the entire body; being hemorrhagic in character, it does not disappear on pressure. It may be preceded or followed by a diffused mottling of the skin or by an erythematous rash. Herpes about the lips or other portions of the face may occur. Bed sores are not uncommon in chronic cases.

Pronounced *nutritional disturbances* occur early and continue throughout the disease. Emaciation and loss of strength are rapid and progressive.

Blood examinations show a marked *polymorphonuclear leukocytosis,* which, according to Huber, varies from 18,000 to 40,000.

**Differential Diagnosis.**—Lumbar puncture enables us to make an accurate diagnosis in this form of meningitis. The cerebrospinal fluid ob-

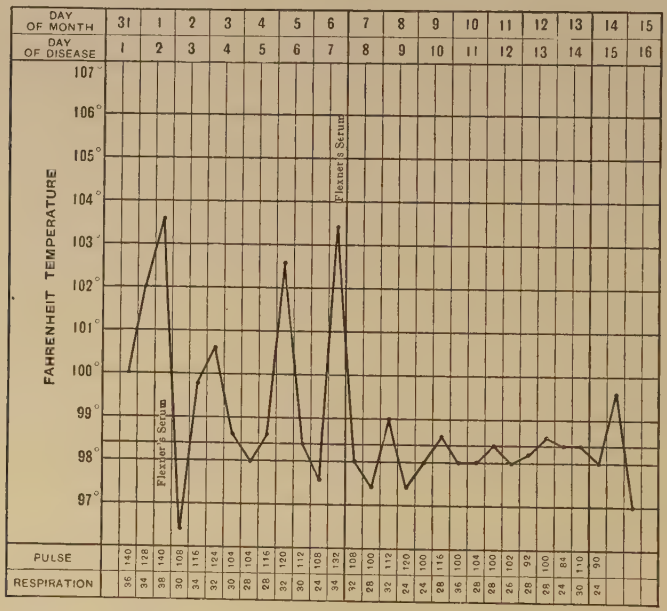

DAY OF MONTH
31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
DAY OF DISEASE
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
FAHRENHEIT TEMPERATURE
107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
Flexner's Serum
Flexner's Serum
PULSE
RESPIRATION

Fig. 96.—Meningococcus Meningitis in Child Six Years Old.

tained early in this disease always contains the diplococcus intracellularis, either free or within the polynuclear leukocytes which predominate in this fluid. They may be demonstrated by microscopical examination of the sediment obtained by centrifuging the fresh cerebrospinal fluid or by examining cultures inoculated with this fluid. The finding of these diplococci establishes beyond a doubt the diagnosis of meningococcus meningitis and, if the technique be accurate, the failure to find these organisms during the first week precludes this diagnosis. As the disease progresses the finding of diplococci in the cerebrospinal fluid becomes slightly less certain, so that after the first week a number of negative findings is necessary to

justify the conclusion that the disease is not meningococcus meningitis. It is quite impossible to make an accurate differential diagnosis between the different forms of meningitis in any other manner than by a careful examination of the cerebrospinal fluid, and yet there are differences in the clinical syndromes of the most common forms of meningitis which, when carefully studied, may materially assist in making the differential diagnosis. The onset in tuberculous meningitis is commonly insidious, and that of meningococcus meningitis is sudden and tumultuous. In meningococcus meningitis the initial high fever, the pronounced hyperesthesia, the high leukocyte count, and the well-marked retraction of the head and posterior spinal curvature are in contrast to the slight fever, the absence of hyperesthesia, the low leukocyte count, the slight retraction of the head, and the tuberculin skin reaction which are commonly present in tuberculous meningitis. By contrasting these symptom groups and carefully inquiring into the previous personal and family history of the patient, and by further considering the fact that in the absence of an epidemic of meningococcus meningitis, the tuberculous is much the most common form of this disease, one can, as a rule, rather definitely differentiate the tuberculous from the meningococcus type (see Tuberculous Meningitis).

**Complications.**—Conjunctivitis, otitis, and pneumonia are the most common complications.

**Sequelæ.**—In cases not treated with the Flexner serum from 20 to 30 per cent. of those that recover have more or less serious sequelæ. The most dreaded after-effects are deafness and mental deficiency. Chronic hydrocephalus, blindness, and various forms of paralysis (cerebral, spinal, and peripheral) may also result. Under the influence of the Flexner serum, in the cases that recover, the cure is usually complete; rarely, permanent deafness may result. The joint affections, which sometimes result, can, as Ladd and Netter have shown, be favorably influenced by direct injection into the inflamed part, of the antimeningitis serum.

**Course and Duration.**—Malignant cases may run a very rapid course with wild delirium, coma, and opisthotonos, ending fatally in a few days. In the great majority, however, the ordinary course of the disease is protracted, lasting for weeks, and in the chronic cases for months. In those cases that recover convalescence is slow, many months being required to restore the child to its normal condition. Under the Flexner serum the course of the disease is materially modified, its duration shortened, and convalescence is much more rapid and complete. In these cases the average duration is now believed to be less than two weeks. A marked improvement in the patient's general condition is often seen within twenty-four hours, although rigidity of the neck and Kernig's sign may persist for many days.

**Prognosis.**—The younger the child the greater the mortality. Very young infants not treated with the Flexner serum rarely recover. Sporadic cases are commonly milder in type than those that occur during epidemics. The general mortality in the past has varied from 70 to 85 per cent. The

indications now are that under the serum treatment this mortality will be reduced to 25 or 30 per cent.

**Prophylaxis.**—In the light of our present knowledge of the etiology of the disease, and from the fact that a number of persons in the same family are not infrequently affected, it is obligatory for the physician to assume its contagiousness. The sick should be isolated from the well and all catarrhal discharges from the throat and nose of the patient should be disinfected. The cerebrospinal fluid drawn by lumbar puncture should be so handled that it may not infect the surroundings of the patient, and all individuals coming in contact with the sick should wash their throats and nasal passages once or twice a day with an alkaline antiseptic solution.

**Treatment.**—Serum Treatment.—The antimeningitis serum developed by Simon Flexner is now looked upon as the specific treatment for this disease. Its use has been followed by remarkably curative results. It is produced in the horse by inoculation of the animal with the diplococcus intracellularis and its products. In suspicious cases if the cerebrospinal fluid is cloudy, the serum is to be immediately introduced into the cerebrospinal canal. Subsequent doses are to be given if the diplococcus intracellularis is demonstrated in the cerebrospinal fluid, or if the first dose is followed by apparent improvement in the symptom group.

*Technique of Administration.*—The serum is injected into the spinal canal. It is furnished in bottles containing 15 c. c. and is stored at refrigerator temperature, so that great care must be taken to have the serum warmed to body temperature before it is injected. Lumbar puncture is made under strict aseptic precautions, and all of the cerebrospinal fluid that will run freely is carefully collected and measured. When the flow has stopped, a syringe containing an amount of serum about equal to the amount of cerebrospinal fluid that has escaped is attached to the same needle, and through it the contents of the syringe are slowly injected into the spinal canal. The fluid should flow from the syringe without resistance; forcing the fluid into the canal under pressure is fraught with danger.

*Dose.*—The average initial dose is 30 to 40 c. c. In very severe cases and in older children this dose may be increased to 45 c. c., provided this amount of serum can be injected without undue pressure; occasionally a quantity of serum greater than the amount of cerebrospinal fluid withdrawn may be injected without resistance. It is commonly necessary to inject 30 c. c. of the serum daily for four or five days, and this routine is followed in the treatment of the average case, unless the symptoms quickly disappear under the treatment. In the event of a quick response to treatment the interval between the injections may be two or three days, and they may be discontinued when convalescence is apparently established and the diplococci have disappeared from the cerebrospinal fluid. If relapses occur and diplococci reappear in the cerebrospinal fluid of cases that have been apparently convalescent, the serum treatment is to be begun again. The number of doses and the frequency of the dose can be determined only by the manner in which the disease responds to the treatment. As

long as symptoms are present, and diplococci are found in the spinal fluid, the serum injections are to be continued at intervals depending upon the severity of the symptom group.

*Results Obtained.*—Flexner furnishes the following table of cases treated with antimeningitis serum:

"Table of Cases of Epidemic Cerebrospinal Meningitis Treated with the Antimeningitis Serum.

*Cases Analyzed According to Age Groups.*

| Age Years. | Total No. Cases. | Recovered. | Died. | Per cent. Mortality. |
|---|---|---|---|---|
| 1-2 | 104 | 60 | 44 | 42.3 |
| 2-5 | 112 | 82 | 30 | 26.7 |
| 5-10 | 113 | 95 | 18 | 15.9 |
| 10-15 | 101 | 73 | 28 | 27.7 |
| 15-20 | 107 | 72 | 35 | 32.7 |
| 20+ | 175 | 106 | 69 | 39.4 |
| Total, all ages | 712 | 488 | 224 | 31.4 |

"This table brings out several points of interest. The highest mortality is shown to have occurred in the first two years of life. But, contrary to the rule under the older forms of treatment in which the mortality was 90 per cent., or over, in this series it was 43.3 per cent. The second age period is from 2 to 5 years, in which the mortality was 26.7 per cent. The third age period embraces children from 5 to 10 years of age and gave the lowest mortality of all, namely, 15.9 per cent. The next period extends from 10 to 15 years and gave a mortality of 27.7 per cent. The next period of from 15 to 20 years showed a considerable rise in mortality, equaling 32.7 per cent., and the last period, embracing the cases of 20 years and over, gave a mortality of 39.4 per cent. The average mortality in all the age periods was 31.4 per cent."

The following table from the same source shows the great advantage to be obtained by the early use of the serum and impresses the importance of early diagnosis and early serum treatment:

| Period of Injection of Serum. | Cases | Recovered | Died | Per Cent. |
|---|---|---|---|---|
| First to third day | 123 | 107 | 16 | 16.5 " " |
| Fourth to seventh day | 126 | 96 | 30 | 23.8 " " |
| Later than seventh day | 112 | 73 | 39 | 35. " " |

The specific action of the serum is also shown by the fact that in 25 to 30 per cent. of the cases treated, the disease terminated by a crisis following the injection of the serum.

*Mode of Action.*—The serum is bacteriolytic and but feebly antitoxic. It acts by destroying the diplococcus. This diplococcus furnishes no extra cellular toxins. Its toxins are entirely intracellular and are liberated only by the disintegration of the diplococci. The serum not only destroys the diplo-

cocci but stimulates phagocytosis and thereby causes the dead cocci to be quickly swallowed up by the leukocytes, wherein their intracellular poison is destroyed. The serum, therefore, acts directly by destroying the diplococci and indirectly by stimulating the leukocytes to destroy their toxins. Under the serum treatment the turbidity of the cerebrospinal fluid gradually disappears, and recovery, when it occurs, is more complete, the sequelæ in these cases being comparatively rare. Deafness occurred in a few instances, and this was "the only persistent defect noted." The duration of the disease under the serum treatment was greatly shortened. The average duration of 228 cases was eleven days.

Symptomatic Treatment.—The pressure symptoms are somewhat relieved by the withdrawal of the cerebrospinal fluid; for this reason it is important to allow all of this fluid to escape that will. The dietetic treatment of these cases is most important, as emaciation and loss of strength are rapid. Easily digested food in concentrated form, and alcohol in the form of whiskey or brandy well diluted, should be given; in young infants the nutritional problem is very difficult. The patient's surroundings should be as quiet as possible, and the room darkened so that he may not be irritated by noise and light. An ice-bag should be applied to the head if it does not worry the patient. Warm baths followed by gentle rubbing of the skin with alcohol may be of service in promoting the peripheral circulation and preventing bedsores; these measures, however, can do more harm than good during the stage of acute hyperesthesia of the skin. Chloral and veronal may be used to produce sleep. In the milder cases the bromids, and in the more severe cases the opiates, may be called for. Opium should not be used unless it be necessary, but the severe pains and nervous irritability which are present in some cases may demand the hypodermic use of morphin; if so, the initial dose should be small, 1/20 to 1/50 of a grain, and gradually increased, if necessary. Tincture of strophanthus or tincture of digitalis may be used to stimulate the heart's action, and collapse may be combated by the subcutaneous injection of normal salt solution or camphor dissolved in sterile olive oil. During the slow convalescence which occurs iodid of potassium may be given. Before the days of serum treatment this drug was largely used and was believed to exercise a favorable influence in removing inflammatory exudates.

## PURULENT MENINGITIS

The term *purulent meningitis* is used to include all forms of meningitis not produced by the tubercle bacillus or the diplococcus intracellularis (meningococcus). It is, therefore, from an etiological standpoint not a distinct disease, but a pathological condition which may be caused by a number of pathogenic microorganisms. The clinical picture produced in these cases, regardless of the specific etiological factor, is so similar that for clinical reasons one is perhaps justified in grouping these various forms of meningitis under the same clinical heading. The term "purulent,"

however, as applied to this group of cases is misleading, in that it implies that tuberculous and meningococcus meningitis are not purulent, while in both of these forms the formation of pus may be a part of the pathological process.

The most common causes of purulent meningitis are the pneumococcus, the streptococcus pyogenes, the bacillus influenzæ, the staphylococcus pyogenes, the typhoid bacillus and the colon bacillus. Of these the pneumococcus is by far the most common.

**Symptomatology.**—The onset and general symptomatology of purulent meningitis more closely resemble the meningococcus than the tuberculous form. Its symptoms are frequently masked by the primary disease of which it is a complication; this is especially true when it is secondary to pneumonia, erysipelas and septicemia. In these conditions a meningitis involving the convexity of the brain may develop without immediately adding to the existing symptom group, other than to increase the delirium and deepen the stupor into a coma. However, in the great majority of cases of purulent meningitis, the characteristic symptoms of meningitis, as previously detailed, are sudden in onset and violent in character. One usually sees a sudden rise of temperature, projectile vomiting, marked general irritation, convulsions, rigidity and retraction of the neck, photophobia, contracted and unequal pupils, rapid, and later irregular, pulse, irregular and sighing respirations, the tâche cérébrale, Kernig's sign, loss of consciousness and profound coma; localized convulsions and contractures may occur. The disease in the great majority of instances ends fatally within a week or ten days from the onset of the initial symptoms.

**Diagnosis.**—The differential diagnosis of the various forms of purulent meningitis can be made only by a careful examination of the cerebrospinal fluid (see Tuberculous Meningitis). A careful examination of this fluid will almost invariably reveal the exciting cause, and, with the finding of the pneumococcus, the streptococcus, the bacillus influenzæ, the staphylococcus, the typhoid or the colon bacillus, the differential diagnosis is definitely made. The differentiation of purulent meningitis from the common tuberculous form of this disease, as based upon the clinical syndromes produced, has already been noted under Tuberculous Meningitis.

Pneumococcus meningitis is by far the most common form of purulent meningitis and is perhaps always associated with a general pneumococcic infection, the pneumococci being present in the blood as well as in the cerebrospinal fluid. It is usually associated with pneumonia or bronchitis. It is sudden in its onset, and, as a rule, runs a short and violent course, always terminating in death. The duration of the disease is commonly from three to eight days.

Streptococcus and staphylococcus meningitis may be rapid or gradual in their onset. They may occur as complications of erysipelas, septicopyemia, middle-ear disease, mastoiditis, and fractures of the bones of the skull, as a complication in spina bifida and as a mixed infection in tuberculous meningitis. These cases run a somewhat less violent course

than the pneumococcic cases; the streptococcus form always terminates in death; the staphylococcus cases show a slight percentage of recoveries.

Influenza meningitis is, as a rule, less sudden in its onset, but violent and characteristic symptoms of meningitis soon develop, and the disease, in the great majority of cases, ends fatally. The percentage of recoveries here, however, is greater than in the preceding forms.

Typhoid meningitis occurs as a complication of typhoid fever. It is commonly fatal, but less so than the other forms of purulent meningitis.

**Treatment.**—Antimeningitis serum is of absolutely no value in the treatment of the forms of purulent meningitis above noted. In these cases, however, it is impossible to make a differential diagnosis of the exact type of

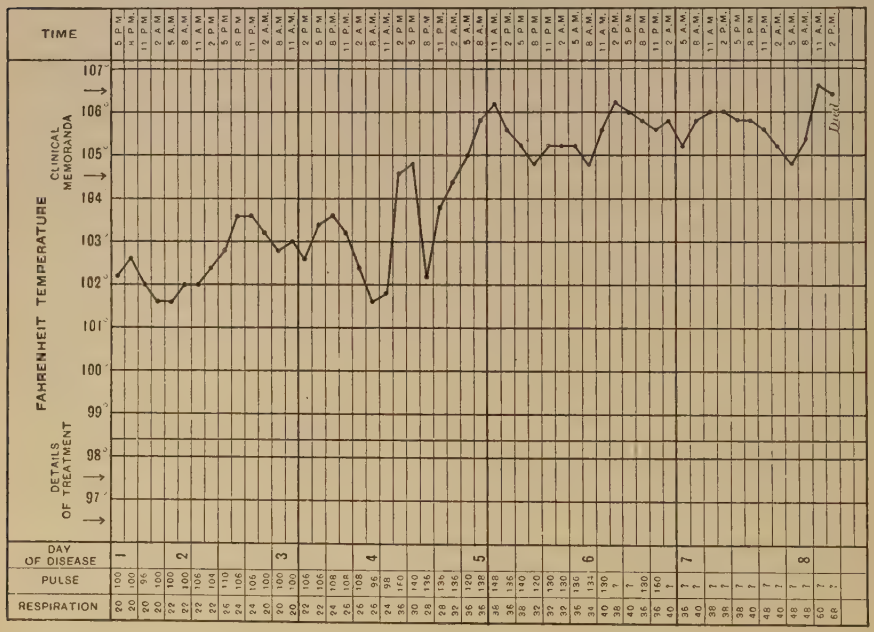



Fig. 97.—Typical Case of Pneumococcus Meningitis; Child Ten Years of Age.

meningitis until a careful examination of the cerebrospinal fluid has been made. As this examination necessitates the loss of valuable time, and as the early administration of the antimeningitis serum is of so much importance in the meningococcic cases, it is perhaps advisable in all cases of purulent meningitis, where the diagnosis is in doubt, to inject into the spinal canal 30 c. c. of antimeningitis serum when the first lumbar puncture is made. If the examination of the fluid thus withdrawn shows the disease to be due to some other organism than the diplococcus intracellularis, the use of the antimeningitis serum is to be discontinued and the disease is to be treated symptomatically as outlined under Meningococcus Meningitis.

Early and repeated lumbar puncture is advisable in all forms of meningitis. It relieves the pressure symptoms, and perhaps in the influenzal, typhoidal, and staphylococcus forms of meningitis it may increase the faint chances of recovery.

Homologous vaccines, if given early, may be of value and should, therefore, always be used in the staphylococcus form.

The symptomatic treatment of purulent meningitis should include absolute rest and quiet in a well-ventilated room, careful and, if necessary, forced feeding, cathartic medication, ice-bags to the head, chloral and bromids to relieve nervous symptoms, and in some cases small doses of morphin hypodermically, to control pain and convulsions. If the internal ear, the mastoid, or frontal sinuses are infected, surgical interference may be resorted to to drain these cavities, but it is questionable whether surgical measures of this kind are ever of any real advantage after the symptoms of meningitis have appeared.

## CHAPTER LXXX

### DISEASES OF THE SPINAL CORD

### ACUTE ANTERIOR POLIOMYELITIS

(*Infantile Spinal Paralysis, Polioencephalitis*)

Acute anterior poliomyelitis is an acute infectious, slightly contagious disease occurring both epidemically and sporadically. The inflammatory lesions begin in the meninges and spread to the cord, involving especially the gray matter of its anterior horns, but any portion of the central nervous system may be involved. The symptom group produced will depend upon the extent of the anatomical lesions. In nearly all sporadic cases, and in the majority of cases occurring during epidemics, the clinical picture presented depends upon the involvement of the anterior horns and adjacent meninges of the cord, but in the epidemic form many cases occur presenting symptoms of acute polioencephalitis, bulbar paralysis or meningitis.

**Etiology.**—The course and onset of this disease, the not uncommon occurrence of a number of cases in the same family, and its appearance in epidemic form all point to the now generally accepted conclusion that it is caused by a specific microorganism, but as yet this microorganism has eluded the most careful search of bacteriologists. Landsteiner and Popper transmitted poliomyelitis to monkeys by inoculating them with an emulsion of the spinal cord, taken from a child just after death from this disease. Flexner and Lewis succeeded in carrying the strain of the virus thus obtained through many generations of monkeys. The virus is not destroyed by glycerination, will pass through the finest filters, is injured by heat, 45° to 50°C., but is not destroyed by drying or by cold; it "belongs to the

class of minute and filterable viruses that have not thus far been demonstrated under the microscope." The disease may also be transmitted by the brain substance, lymph nodes, salivary glands, tonsils, mucous membrane of the nasopharynx, and by the blood and cerebrospinal fluid of monkeys ill of this disease. The experiments of many observers have demonstrated that the virus from the above-named sources will produce the disease in monkeys when injected into the brain, the spinal cord, the tissues adjacent to peripheral nerves, the blood stream, and the anterior chamber of the eye, and also by rubbing the virus into the mucous membrane of the nasopharynx or introducing it into the stomach or intestines. These experiments suggest that infection in human beings may occur through the nasopharynx or gastrointestinal canal. The manner in which the virus may be transmitted from one individual to another is not known. In its epidemic form the disease extends through low-lying, well-watered areas and along routes of travel. It may be spread by abortive ambulant cases, by healthy intermediate carriers, by animals having the disease, and perhaps by insects such as the common house-fly, and by fomites such as dust.

Acute anterior poliomyelitis may occur sporadically or in epidemic form. The most notable epidemic in this country occurred in New York City and its surroundings in 1907; it included 2,500 cases. Holt and Bartlett made a very complete report on the "Epidemiology of Acute Poliomyelitis," which included an analysis of thirty-five epidemics. They found "many instances of closely connected groups of cases. In one instance there were seven in one family; in three instances four in a family; five instances of three in a family, and in all forty instances, comprising 69 cases of more than one in a family." The startling increase in the epidemic form of this disease is shown in Lovett's compilation of the reported epidemics since 1881:

| Years. | Cases. | Outbreaks. | Average Number of Cases. |
|---|---|---|---|
| 1880-1884 | 23 | 2 | 11.5 |
| 1885-1889 | 93 | 7 | 13 |
| 1890-1894 | 151 | 4 | 38 |
| 1895-1899 | 345 | 23 | 15 |
| 1900-1904 | 349 | 9 | 39 |
| 1905-1909 | 8,054 | 25 | 322 |

The sporadic form of this disease is rare during the first few months of life, but in the latter half of the first year it is quite common; the second year of life is the period of greatest susceptibility, and in the third year it is still quite frequent, but after the fifth year it is comparatively rare; about 50 per cent. occur during the first and second years, and 80 per cent. during the first three years.

The epidemic form occurs with almost equal frequency in young childhood as in infancy; it is very common between the ages of four and thirteen, and not uncommon in early adult life. This form is also slightly transmissible by direct contact; the degree of contagiousness is not very

great, since only a small proportion of exposed individuals contract the disease. It occurs with slightly greater frequency in boys than in girls. Dry, warm weather very materially influences its spread. Fifty per cent. of the cases occur during the dry hot months of August and September, and 70 or 80 per cent. between the first of June and the first of October. Epidemics occur almost invariably during the warm, dry months, and are usually terminated by cold weather; occasionally, however, an epidemic is continued with sporadic outbreaks after cold weather has begun.

**Pathology.**—Acute anterior poliomyelitis is a general infection producing especially an interstitial inflammation characterized by congestion, perivascular round-celled infiltration and edema of the leptomeninges, the spinal cord and brain. Inflammatory changes occur first in the pia mater of the cord and the medulla, and are most marked around the blood vessels; about these the round-celled infiltration is greatest and may contract their lumen, producing congestion, edema and minute hemorrhages.

The cerebrospinal fluid is increased in quantity and quite early in the disease is opalescent, due chiefly to an increase in the number of lymphocytes, although polymorphonuclear leukocytes may also be present. By the third or fourth day the cerebrospinal fluid is clear, but is still increased in quantity, and contains a large number of lymphocytes.

All of the blood vessels of the cord coming from the inflamed pia are congested and show perivascular round-celled infiltration, and there results a more or less general myelitis, more marked in the gray matter of the anterior horns and commonly most severe in the cervical and lumbar enlargements than in other segments of the cord. This inflammation in the cord, like that in the meninges, is marked by congestion, round-celled infiltration, edema and minute hemorrhages, and results in extensive damage to nerve cells, especially the motor nerve cells in the anterior cornua. Ganglion cells, however, in the posterior horns, especially in Clark's column, are not uncommonly affected. The degenerative changes in the nerve cells may result in their complete destruction, and a permanent motor paralysis and atrophy of the muscles supplied by the axons coming from these cells. Edema, or temporary alteration leading to functional impairment, but stopping short of permanent degeneration of the ganglion cells, may cause a transient paralysis of the muscles innervated by their neurons. The predominance of these lesser lesions explains the fact that the greater part of the widespread paralysis occurring during acute anterior poliomyelitis is transient, and also explains the hopeless character of the final permanent paralysis of an associated muscle group.

It is important to remember that inflammatory lesions may also occur in the white matter of the cord, thereby implicating the long ascending and descending tracts, thus explaining such rare lasting motor disturbances as ataxia, exaggerated reflexes and muscular spasticity. Inflammatory changes in the spinal ganglia, the medulla oblongata and pons varolii, similar to those above noted in the cord, may occur, and infiltration around the deep nuclei of cranial nerves explains the facial and other

paralyses of these nerves. Like changes may occur in the cerebellum and less rarely in the cerebral cortex. In fatal cases not only the pia mater and the spinal cord, but the medulla, pons, basal ganglia and even the cerebrum may be involved in the inflammation. In mild cases, especially of the sporadic type, the inflammatory changes are almost or quite limited to the anterior half of the cord, and the paralysis which accompanies and follows this condition is largely due to degenerative changes in the anterior horn cells of its cervical and lumbar enlargements.

Chronic Changes.—As time goes on many of the injured ganglion cells entirely recover, others are destroyed, and the axons emanating from them disappear. The destruction of the nerve roots makes the anterior nerve root bundles smaller and produces an atrophy of the muscles, while the absence of the trophic influence of the anterior cornual cells is shown by more or less lack of development of that part of the body supplied by the diseased nerve cells. In such cases also the affected part of the cord is diminished in size and sclerotic changes occur.

Lesions are found outside of the nervous system, but they are not characteristic. Bronchopneumonia and parenchymatous degeneration of the liver and kidneys occur. Changes in the mucous membrane of the small intestine and stomach are common; congestion and enlargement of the solitary follicles, Peyer's patches and mesentery glands are common.

**Immunity.**—One attack is believed to confer immunity.

**Symptomatology.**—Wickman has described eight distinct types of acute anterior poliomyelitis, namely: the spinal poliomyelitic; the abortive; the ascending or descending; the bulbar or pontine; the encephalitic; the ataxic; the polyneuritic, and the meningeal.

Spinal Poliomyelitic Type.—This is the ordinary form. Its onset is marked by nervous irritability, restlessness, headache, pain in the spine and extremities, fever, sweating, marked prostration, and gastrointestinal disturbances. Vomiting is common, diarrhea is present in about one-half the cases, and constipation in the remainder. Tonsillitis may also occur as an initial symptom. The fever in mild cases varies from 100° to 101°F.; in severe cases it may reach 105°F. It occurs as an early symptom and continues in mild cases for one or two days, and in severe cases for a week. Hyperesthesia is present, and movement of the body, especially the head, produces pain. The pain, tenderness, and rigidity of the muscles of the neck and spine are early and characteristic symptoms, usually associated with a slight retraction of the head. The mind is usually clear, but apathy and drowsiness may occur; very rarely delirium, convulsions, and coma are seen. Early, the deep reflexes are frequently exaggerated, but later they are diminished and vasomotor disturbances are very common. Paresthesia or numbness may precede or accompany the beginning paralysis. Meningitic symptoms are present in some cases; with the pain, stiffness, and tenderness of the neck and spine there may be a marked retraction of the head, intense nervous excitability, photophobia and a mod-

ified Kernig's sign. The spleen is enlarged and leukopenia with a relative increase of lymphocytes occurs early.

The clinical syndrome made up in whole or part of the symptoms above noted may continue for four or five days, and then gradually subside, and during this time the characteristic paralysis makes its appearance. With the onset of this paralysis the pain, tenderness, and hyperesthesia previously noted are increased in the paralyzed part, and, rarely, a marked line of tenderness may be noted along the peripheral nerves. In some cases, especially the sporadic ones, the fever and acute symptoms above detailed may be slight or absent, and the typical paralysis may develop with a few accompanying symptoms; such cases are usually mild, and the paralysis is not widely distributed.

Abortive Type.—During epidemics it is estimated by various observers that from 15 to 50 per cent. of all cases belong to the so-called abortive type. They present a syndrome which commonly includes the following symptoms: fever, headache, nausea, vomiting, diarrhea, constipation, nervous irritability, pain in the neck, back and limbs, ataxia, diplopia, and exaggerated or diminished patellar reflex, but which may include any of the above-named symptoms associated with the ordinary form of this disease. This symptom group, however, subsides into convalescence without a supervening paralysis, but these cases usually show extreme weariness and muscular weakness. Their diagnosis largely depends upon the fact that they occur during epidemics of acute anterior poliomyelitis and resemble in their onset, and in many of their symptoms, other cases of this disease having the supervening paralysis. The immune bodies which are present in the blood of patients recently recovered from frank attacks of this disease can also be demonstrated in the blood of these abortive cases by the fact that they render inactive the specific virus of this disease.

The Ascending or Descending Type.—The clinical course of this type is identical with that of so-called Landry's paralysis. It may appear first in the lower or upper extremities or in the muscles supplied by the cranial nerves; it then descends or ascends. The ascending type is more common; it begins in the legs and may involve almost the entire body. As it progresses upward the muscles of the legs, abdomen, back, chest, arms, neck and diaphragm may be involved, and death commonly ensues from paralysis of the external muscles of respiration or from phrenic paralysis within from one to three days. This form must not be confused with that in which death results from paralysis of the centers of respiration.

Bulbar or Pontine Type.—During epidemics, cases not infrequently occur in which the severe constitutional symptoms of acute polioencephalitis are associated with paralysis of the cranial nerves whose nuclei are situated in the medulla oblongata and pons cerebri. The paralysis of these nerves may or may not be associated with paralysis of the trunk, neck or extremities. The facial paralysis in these cases is commonly unilateral and usually associated with an oculomotor paralysis, causing divergent squint with or without ptosis. Disturbances of deglutition, dyspnea, and

irregular respiratory action, with a rapid and irregular pulse, are common symptoms. The bulbopontine paralysis occurring in these cases may almost or quite disappear, leaving perhaps a slight facial or oculomotor paralysis. In severe cases obstinate constipation and retention of urine may occur, and death may result from splanchnic, cardiac, or respiratory paralysis.

The Encephalitic Type.—This is characterized by symptoms resembling meningitis, and is associated with paralyses due to lesions in the motor areas of the brain, such as spastic monoplegia or hemiplegia, or to paralyses of the bulbar or pontine type.

The Ataxic Type.—This is a polioencephalitis involving also the cerebellum. The symptoms, as in the encephalitic form, are very acute and violent, but the meningeal symptoms are associated with ataxia of

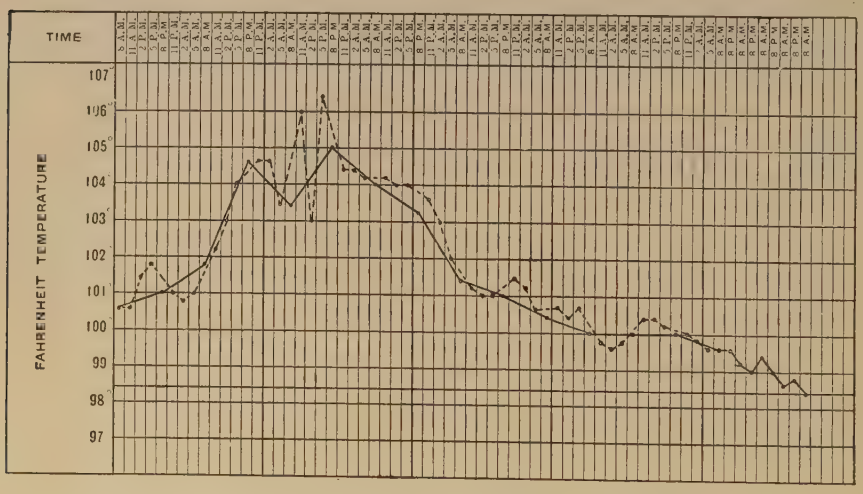



Fig. 98.—Acute Anterior Poliomyelitis of the Bulbar or Pontine Type.

In this case four doses of 16 ounces of normal salt solution, each containing 10 grains of urotropin, were given on the third and fourth day. After this treatment the child made a slow and complete recovery.

movement of the arms and legs, nystagmus, and explosive syllabic speech; this form is very rare.

The Polyneuritic Type.—In this type the peripheral nerves are involved, presenting a picture somewhat resembling multiple neuritis. It is not uncommonly associated with the ordinary spinal poliomyelitic type. Pain is the prominent symptom; it may be commonly elicited by pressure over the nerve trunks or joints of the paralyzed parts. In some cases the pain and tenderness are very marked in only slightly paralyzed parts, so that early in the disease the paralysis may be overlooked and a diagnosis of rheumatism or scurvy made. In other cases there may be a combination of flaccidity and spasticity, the latter resulting in contractures which may cause errors in diagnosis.

The Meningeal Type.—This is an indistinct type, probably covered by

the encephalitic type above noted. It presents the symptoms of meningitis associated with paralyses of various kinds.

Paralysis.—This is the characteristic symptom which confirms the diagnosis. During epidemics it may occur within the first twenty-four hours; in the sporadic form it is not, as a rule, recognized until much later, but in all cases, except those of the abortive type, it becomes so evident between the second and the fifth day that it can scarcely be overlooked, even though it be overshadowed by violent acute symptoms. Anderson & Frost say: "Cases of acute anterior poliomyelitis are encountered showing all gradations in the degree and extent of paralysis. In the same group may be found cases resulting in extensive and lasting paralysis; cases with permanent paralysis of slight extent; cases in which the patients have transient paralysis, recovering completely within a few weeks or even a few days; other cases in which there is no definite paralysis, but merely muscular weakness of short duration; still others in which the only motor disturbance is ataxia, tremor or a transient ocular disturbance, such as diplopia or nystagmus. Finally, within the same group are seen cases of illness exhibiting only the symptoms of a general infection, usually accompanied by symptoms indicative of meningeal, spinal or encephalitic irritation, but without definite motor disturbances." In the majority of instances the paralysis is motor and is complete and flaccid in character; the part involved offers no resistance and falls limply into the position gravity directs. It reaches its maximum in from one to three days after its onset and involves the muscles of one or more limbs, and, more rarely, the muscles of the trunk, neck and face. After a period of one or two weeks the paralysis begins to subside and the patient commences to regain motion in the paralyzed part. This improvement may continue rather rapidly for four or five weeks, leaving, in 90 per cent. of the cases, a group of muscles functionally related, still paralyzed. The extent of the remaining paralysis represents the actual damage to the part involved and indicates the location and extent of the injury to the motor cells of the anterior horn or similar cells in the upper-lying parts of the nervous

Fig. 99.—The Two Commonest Varieties of Foot Deformity from Anterior Poliomyelitis, in the Same Patient.

In the right foot, equino-varus from paralysis of the peroneal group; in the left foot, valgus from paralysis of tibialis anticus and posticus. (A. Freiberg.)

system. After the third or fourth month the amount of improvement which occurs in the paralyzed member depends largely upon the treatment instituted, but, for the most part, the muscular paralysis then existing is permanent. In the great majority of cases the paralysis is confined to the lower extremities, and, as a rule, only one leg is involved. When the upper extremities are involved the paralysis is usually general, both arms and legs being affected, and the trunk and face muscles may also be involved. Less commonly one upper extremity is involved with one lower, usually on opposite sides, and still less frequently one upper and both lower extremities, and very rarely one upper extremity alone is paralyzed.

FIG. 100.—QUADRUPEDAL GAIT IN A BOY OF TEN YEARS RESULTING FROM ANTERIOR POLIOMYELITIS.

There was marked flexion contracture of the right hip and paralysis of the quardriceps extensor and sartorius of the right thigh. Extensive paralysis of leg muscles on both sides. Boy enabled to walk erect without crutches by means of tenotomies and tendon transplantation. (A. Freiberg.)

In the rare cases, where the cerebrum as well as the spinal cord is involved, we may have a combination of flaccid and spastic paralysis.

MUSCULAR ATROPHY is one of the most notable features of this disease. Atrophy and fatty degeneration of the paralyzed muscles and their tendons quickly begin, and become very marked after a few weeks. The wasting of muscles very materially reduces the size of the affected limb. -In extreme cases the circumference of the part may be reduced to almost half its original size; this may in part be due to the loss of subcutaneous fat. The growth of the paralyzed limb is diminished so that in time it may be much shorter than its fellow. In severe cases the wasted, dwarfed and withered extremity is loose-jointed and markedly deformed. These deformities result largely from the action of the normal muscles of the part, the antagonistic muscles being paralyzed. The foot, leg, hand, arm, and spine may thus be drawn into abnormal positions, resulting in various forms of clubfoot, clubhand, curvature of the spine and subluxation of joints, especially the shoulder and knee. The skin of the paralyzed part is

notably reduced in temperature, it appears dry, shriveled, and at times cyanosed, and is cold and lifeless to the touch. A further evidence of the loss of the trophic influence of the anterior cornual cells is the great reduction in the size of the bones of the affected extremities, as can be clearly shown by skiagraphs.

The CHANGED ELECTRICAL REACTIONS of the paralyzed muscles may be a very important diagnostic sign. They fail to respond to the faradic current, and, as the atrophy progresses, they show the reactions of degeneration to the galvanic current; that is to say, they respond feebly and slowly, and the anodal closure contraction exceeds the cathodal closure contraction, and after a time they fail entirely to respond to any form of electrical stimulation.

The reflexes in the paralyzed part are nearly always absent. The knee-jerk, however, may be present where the disease of the cord is confined to the cervical or lower dorsal region, and it may also be present in those cases seen during epidemics where the disease is confined to other portions of the central nervous system than the cord.

**Diagnosis.**—The early stage of acute anterior poliomyelitis so closely resembles that of other acute infections that it may be impossible to make a diagnosis until the paralysis is discovered. During an epidemic, however, the physician should at least make a provisional diagnosis in the presence of the symptom groups which ordinarily announce the onset of this disease. It may be mistaken for acute intestinal infection or influenza, because of the presence of fever, vomiting, and nervous symptoms, but these disorders should soon be excluded by the subsequent course of the disease. When announced with high fever, general hyperesthesia, convulsions, and stupor, it may be mistaken for some form of meningitis; in such cases lumbar puncture may materially assist in clearing the diagnosis, since a bacteriological examination of the cerebrospinal fluid fails to reveal the presence of microorganisms, but shows a large number of polynuclear or mononuclear cells. In the early stages the blood may show a leukopenia and a moderate lymphocytosis, and Gay and Lucas note that the differential count shows a relative increase in the number of eosinophiles.

With the onset of the paralysis there should be little difficulty in making a diagnosis if the following points pertaining to the differential diagnosis of the common paralyses of infancy, viz., infantile paralysis, cerebral palsy, and multiple neuritis, are kept in mind:

| ACUTE ANTERIOR POLIOMYELITIS | CEREBRAL PALSY | MULTIPLE NEURITIS |
|---|---|---|
| Occurs most commonly during second year. | Occurs most commonly during first year. | More common in childhood and adult life. |
| Occurs sporadically and epidemically without apparent cause. | Usually due to cerebral hemorrhage or congenital defects, parturient injuries. | Follows diphtheria, rarely produced by other causes in childhood. |

| Acute Anterior Poliomyelitis | Cerebral Palsy | Multiple Neuritis |
|---|---|---|
| Convulsions, not commonly repeated, occur in 10 to 15 per cent. of the cases. | Repeated convulsions in most cases; frequently a causative factor. | No convulsions. |
| Paralysis (motor) complete, flaccid. Limb limp and non-resistant. | Paralysis (motor) partial, spastic. Limb rigid, contractures present. | Paralysis (both motor and sensory) complete, flaccid. Limb limp, and may be acutely sensitive along the line of the nerve. |
| Paralysis commonly confined to one or both legs. Arms, trunk and back may be involved. | Paralysis, hemiplegic; other forms such as diplegia, paraplegia and monoplegia comparatively rare. | Paralysis, paraplegic, or limited to the distribution of the nerves involved. |
| Muscles atrophied and degenerated after a few weeks. | Muscles not atrophied. | Muscles slightly atrophied, but recover. |
| Limbs shorter from lack of growth, a late symptom. | Lack of development, not so marked. | No shortening. |
| Electrical reactions, altered and lost. | Electrical reactions, normal. | Electrical reactions, altered and diminished, sometimes lost. |
| Knee-jerk commonly lost. | Knee-jerk increased. Babinski sign, perhaps clonus. | General loss of reflexes in part involved. |
| Intellect not involved. | Lack of mental development. Epilepsy may result. | Central nervous system in no way involved. |

**Prognosis.**—Holt and Bartlett found in the epidemic form of this disease a mortality of 12 per cent. in 1,659 cases. Wickman found the death rate under eleven years of age to be 12.2 per cent., and from twelve to thirty-two years of age 27.9 per cent. The Massachusetts State Board of Health found the mortality to be 16 per cent. in infants under one year; 4 per cent. from one to ten years, and 20 per cent. in individuals over this age. In this type of the disease complete recovery without residual paralysis may occur in from 10 to 16 per cent. Death occurs most frequently during the first week, and commonly results from paralysis of respiration. In the sporadic form death is very rare, and complete recovery without sequelæ is very uncommon. Within a few hours such destruction may be wrought among the anterior horn cells that the patient is more or less crippled for life. There is, however, no way of making an accurate prognosis from the early symptoms as to the amount of residual paralysis. The physician is therefore justified in being very guarded in his early prognosis. He may comfort the family by telling them that the intellectual development of the child will in no way be affected, but in predicting the amount of residual paralysis he should remember that there is a possibility that the child may almost, if not completely, recover. After a month or six weeks the extent of the residual paralysis may be approximately estimated by the electrical reactions of the paralyzed muscles; those that fail to respond to any form of electrical stimulation will probably remain paralyzed.

The prognosis, so far as improving the function of the paralyzed part, is not altogether hopeless, even after the fourth month. Up to this time the improvement is progressive and spontaneous, but thereafter improvement depends upon securing the greatest possible functional development of those fibers in the paralyzed muscles which have not been hopelessly damaged, and on the education of adjacent muscles to the partial performance of the functions of the paralyzed muscles.

**Prophylaxis.**—These patients should be isolated and rigidly quarantined, and other members of the family should not be permitted to go to school, or to public gatherings. All members of the household, especially those who must necessarily come in contact with the patient, should use two or three times a day a nasal douche or spray of a 1 per cent. solution of peroxid of hydrogen, and they should be given urotropin in from 2- to 6-grain doses, according to their age, three times a day. The discharges from the throat, nose, kidneys and gastrointestinal canal of the patient should be disposed of in such a manner that his surroundings may not be infected, and that the common housefly or other insects may not carry contagion to other parts of the house. A rigid quarantine should be continued for three weeks, and house disinfection should then be resorted to, as detailed in the chapter on Scarlet Fever. Sprinkling of streets and lawns is also regarded as a wise prophylactic measure.

**Treatment.**—General Treatment.—With the onset of acute symptoms, cold applications to the spine are indicated, but in most cases the diagnosis is not made early enough to give the patient the benefit of this treatment. Absolute quiet and rest in bed should be insisted upon. Calomel in small doses followed by Rochelle salts or castor oil should be given to move the bowels and control intestinal fermentation, and should be followed by liquid and easily digested food. Sedative medication to relieve nervous irritability and sleeplessness may be indicated; bromid of soda or phenacetin may be used for this purpose. Rarely an opiate is necessary to allay pain. In severe cases associated with meningeal symptoms, lumbar puncture may relieve the pressure and slightly modify the symptoms. In these cases hypodermoclysis with normal salt solution may be a life-saving measure. Strychnin and caffein-sodium-benzoate given hypodermically may be of value. Whiskey given by the mouth or by the rectum is valuable in all cases when, for any reason, sufficient food cannot be taken.

There is no specific medical treatment for this disease, but animal experimentation indicates that hexamethylenamin (urotropin) should have some therapeutic value, not only as a prophylactic, but also as a curative, remedy. My experience with this drug in the epidemic of this disease which prevailed in Cincinnati in 1911 has led me to believe that it has some curative value if given early and in large doses. From the onset of the acute symptoms it should be given in from 3- to 10-grain doses, according to the age of the child, every four hours, and this dosage should be continued only until the acute symptoms commence to abate. In one appar-

ently desperate case in which the gastrointestinal symptoms were so violent as to preclude all medication by mouth or rectum for a period of four days, the child's life was saved by hypodermoclyses of physiological salt solution, to each of which was added 10 grains of urotropin.

Treatment of the Paralysis.—When the acute symptoms have subsided and the child's life is no longer in danger, attention should at once be directed to the prevention of contractures and deformities in the paralyzed parts. To accomplish this the paralyzed portion of the body should be maintained in as *nearly a normal position as possible* by the use of pillows, long bags of sawdust, or by light braces, or, if necessary, strapping with adhesive plaster. It is most important that during this early acute stage the paralyzed muscles and their tendons and the ligaments of the joints should not be stretched by allowing the paralyzed part to assume an unnatural position. Apart from this there is little to be done except to feed the patient carefully, give him plenty of fresh air, and shield him in every possible way from influences that might produce nervous irritation. Above all it must be firmly impressed upon the parents and attendants that for at least two weeks, and possibly three, following the disappearance of acute symptoms, the paralyzed part is not to be exercised by massage, electricity, or in any other way. Then follows a long period during which electricity, massage, and active and passive exercise of the paralyzed part will be of great advantage. Electricity promotes the nutrition and keeps up the function of those muscles and muscle fibers which have not been wholly cut off from their communications with the cord. The faradic current may be used for those muscles which respond to its use; other muscles more seriously affected may require the galvanic current to produce a contraction. The home application of the galvanic current, however, cannot, as a rule, be easily brought about, so that, in the great majority of instances, the faradic current alone is used, and this is supplemented by systematic massage. Massage, when administered by a skillful operator, exercises the wasted muscle, increases its circulation and promotes its nutrition, and is perhaps more generally applicable in producing these results than is electricity; it should be remembered, however, that in beginning this treatment the weak and wasted muscles require very gentle massage; strong massage may further weaken and injure them. As the child is brought gradually under treatment the most satisfactory results are obtained by the daily use of mild systematic massage combined with the daily use of the faradic current, the one being given in the morning and the other in the afternoon. This routine treatment may be continued daily for three or four weeks, and then for a long period the massage and electricity may be given on alternate days. As soon as the child has recovered sufficiently to use the paralyzed part great good may result from mechanical appliances properly adjusted by an orthopedic surgeon; great harm, however, may follow the unskillful use of heavy braces; the proper use of these appliances should assist the child in the voluntary use of weak muscles without aggravating the deformity, which has probably

already resulted from the unbalanced contraction of muscles. In the late treatment of these cases certain operative measures are of value in relieving deformities and improving muscular action. By lengthening, shortening, anastomosing, or transplanting tendons, muscular action may be utilized which was previously wasted, and loose joints may be made much more serviceable by fixing them by ankylosis. Muscle training is a very important part of the treatment; it should accompany the massage after the third or fourth week of the child's illness, and should follow the operative measures above referred to in the subacute and chronic cases. The patient, stripped and lying on a table, should be taught to make movements which will exercise and thereby develop partially wasted muscles; in the beginning it may be necessary for the instructor to assist the child in making these movements. Exercises of this character may be given for half an hour every day or every second day until the child is able to give up his braces.

## MYELITIS

Myelitis is an inflammation of the spinal cord resulting in more or less disintegration of its tissues. The white matter may be indistinguishable from the gray, and both may present the appearance of red softening. Capillary hemorrhages occur and the anterior horn cells of the affected areas are degenerated. If the destructive lesion be localized to a section of the cord, producing a more or less complete transverse myelitis, the sensory fibers of the posterior and cerebellar columns degenerate upward and the motor fibers of the pyramidal tracts degenerate downward. The transverse forms of myelitis most frequently occur in the upper half of the cord; the dorsal and cervical regions are favorite sites. In disseminated myelitis, resulting from syphilis and other causes, small areas of degeneration may be scattered throughout the cord.

**Etiology.**—Myelitis is produced by bacterial infection. In childhood it occurs most commonly as a manifestation of syphilis, or as a complication of tuberculosis of the spine (Pott's disease); in this latter condition there is a preliminary compression followed by inflammation. It may occur as a sequel of one of the acute infectious diseases or as one of the manifestations of a septic process located anywhere in the body. "Cold" and rheumatism are classed among the exciting causes. Myelitis may result from the extension downward and inward of meningitis (meningomyelitis), and may be produced by injury to the spine, new growths pressing on the cord, and by spinal hemorrhage, but whatever may be the exciting factor the most destructive lesions are inflammatory and are produced by microorganisms, most commonly streptococci and staphylococci.

**Symptomatology.**—Transverse Form.—Fever and constitutional symptoms exist in all forms of myelitis. They are much more marked, however, in the acute varieties due to infection. In these cases we may have a sudden onset, temperature rising to 103° or 104°F., with more or less severe

pain and tenderness over the spine, and within twenty-four or thirty-six hours the characteristic paralysis may begin to develop. These cases are in marked contrast to the much more common ones produced by Pott's disease and syphilis, in both of which the onset is insidious and the febrile reaction slight.

To clearly understand the symptom group presented by myelitis, one should remember that the character and severity of the paralysis will depend upon the location and the severity of the lesion in the cord. In myelitis one finds, as a rule, two distinct kinds of paralysis. In one part of the body the paralysis will be *flaccid,* having all the characteristics of this type as described under Acute Anterior Poliomyelitis. In another and lower part of the body the paralysis will be *spastic,* having all the characteristics of this type as described under Cerebral Palsies. Where the lesion is located in the cervical portion of the cord, the muscles of the arm which are directly innervated by the anterior cornual cells of this part of the cord will be in a state of flaccid paralysis, with loss of reflexes in the part, and sooner or later atrophy and the reaction of degeneration will be marked in the paralyzed muscles. All that part of the body below the lesion in the cervical cord will be in a state of spastic paralysis, which can be most easily recognized in the legs. The leg muscles may even be contractured, the reflexes are exaggerated, but there is no atrophy or reaction of degeneration in the paralyzed muscles. The spastic paralysis is due in these cases to the descending degeneration of the fibers of the pyramidal tract, which begins at the point of the cord lesion and extends downward. If the lesion be in the dorsal portion of the cord, as it commonly is, the arms, and all of that part of the body above the lesion, will be free from paralysis, and the muscles of the trunk directly supplied by nerve fibers from the diseased portion of the cord will be in a state of flaccid paralysis, while the legs will be in a state of spastic paralysis. If the lumbar segment of the cord be involved in the myelitis, there will then be a flaccid paralysis of the lower extremities, with loss of knee-jerk and other reflexes, since the cells in the lumbar cord are in direct communication with the muscles of the legs. In transverse myelitis the paralysis is symmetrical, bilateral, motor and sensory. The upper line of the paralysis is sharply limited by the lesion in the cord and is marked by a small zone of hyperesthesia, sometimes associated with a sensation of belt-like constriction around the body. Immediately below this zone the various forms of sensation are almost or totally lost; complete anesthesia may exist.

Vesical and rectal disturbances are among the characteristic symptoms of myelitis; the urine is retained and dribbles away from an overfull bladder, and there is involuntary discharge of feces. The character, however, of this disturbance of bladder and rectum differs with the localization.

Bedsores very commonly develop, due to trophic disturbances of the skin, which make it possible for slight pressure and irritating discharges to produce extensive sloughing. It is a matter of the greatest difficulty

in these cases to so care for the patient as to prevent the formation of bedsores.

The above clinical picture is that ordinarily presented by acute myelitis of the transverse variety, but it should be remembered that it may be very greatly modified in individual cases, and the modification of the symptoms will depend upon the location, character and extent of the lesions in the cord.

Disseminated Forms.—*Tuberculous myelitis* due to caries of the spine is the most common form of this disease in childhood; in the beginning it is purely a compression myelitis, and the paralysis develops very slowly. During this early stage the sharp lancinating pains radiating from the spine are due to compression of the nerve roots. This is an early and prominent symptom and is associated with marked tenderness over the spinous processes. The early paralysis that develops in these cases is spastic, unassociated with the flaccid variety; it occurs most commonly in the legs, but may occur in the arms. The reflexes are exaggerated, and hyperesthesia rather than anesthesia is present. As the inflammatory process invades the cord unilateral symptoms may appear, but after a time these are replaced by the symptom group above given of ordinary transverse myelitis.

*Syphilitic myelitis* is slow and irregular in its onset; the paralysis may not be symmetrical, is never so well marked, and in this form of the disease the distribution of the paralysis, conforming above to the flaccid and below to the spastic type, is not seen. The morbid process is distributed over a great portion of the cord and is not so intense at any one level, so that there may be great variations in the symptom group produced. Sensory paralysis may not always be associated with motor, and apparent recovery followed by relapses may occur. The irregularity of the symptom group in this form of the disease makes it necessary for one to depend largely upon other evidences of specific disease in making the diagnosis.

**Prognosis.**—In compression myelitis due to Pott's disease the prognosis, so far as recovery from the paralysis is concerned, is rather favorable if the diagnosis is made early and proper treatment is instituted before the cord has become infected; from 50 to 60 per cent. of these cases recover. In neglected cases, where infection of the cord has taken place and a complete transverse myelitis has resulted, the prognosis is bad. In syphilitic myelitis the prognosis is good if an early diagnosis is made and proper treatment is instituted. The symptoms, as a rule, yield to antisyphilitic treatment. In syphilitic cases of long standing, however, while some improvement may follow the treatment, the prognosis, so far as ultimate recovery is concerned, is bad. In acute infectious myelitis the prognosis will depend upon the location and severity of the lesion in the cord. The higher the lesion the greater the danger. The more destructive the lesion, as indicated by the severity of the symptoms, the more serious the prognosis. While on the whole the prognosis in severe lesions located high in the cord is bad, it is not wise to make an early unfavorable prognosis, since it is quite impossible for the physician to foretell with accuracy

the course that the disease will take in an individual case. One may generalize and say, after observing the case for two weeks, that this being a mild case it will probably recover, or this being a severe one it will probably die; yet it should always be remembered that in a few of the severe cases complete recovery takes place. In those cases in which the paralysis persists for months the great danger lies in the complications, such as bedsores, cystitis and sepsis. These are important factors in producing a fatal termination in a large percentage of the subacute and chronic cases.

**Treatment.**—Rest in bed under the most careful nursing, directed especially toward the prevention of bedsores and cystitis, is a most important part of the treatment. The mattress upon which the patient rests should be most carefully selected with reference to conforming to the surface of his body without producing undue pressure at any point. Air and water mattresses are well adapted to this purpose. From the beginning the nurse should be carefully instructed to shift the patient, when awake, every hour or two, so as to avoid long pressure upon any one part of the skin. The skin should be rubbed with alcohol, and, if slight redness occurs, with zinc ointment, and at the first indication of a developing bedsore all pressure should be removed from the part, and every effort made to restore the skin to its normal condition. The retention of urine, which occurs in this disease, necessitates the use of the catheter, so that it is most important in the beginning that the nurse should be carefully instructed to always use sterile catheters, anointed with sterile vaselin, for only in this way may cystitis be prevented. This complication is a much dreaded one, and, in the event that infection of the bladder occurs, it should be carefully washed out two or three times a day with an alkaline antiseptic solution. The dribbling of urine, which occurs in this disease, is a source of danger and irritation, and the patient should be protected from it, if possible, by the proper use of cotton. The nutrition of the patient must be carefully kept up throughout the whole course of the disease by a carefully selected diet, and attention to the digestive organs. Medication that interferes with the appetite or digestion will do more harm than good.

In *myelitis due to Pott's disease* the aim of the physician should be to remove the pressure from the cord and cure the tuberculosis of the spine. To remove the pressure the patient should for a time be placed in bed and kept absolutely at rest. This is to be followed by a plaster-of-Paris jacket, so applied as to relieve the pressure on the cord and partially separate and prevent the rubbing together of the diseased spinal vertebræ. With the jacket properly applied the patient can move and be moved without injury either to the cord or the spine. As the patient progresses the plaster jacket may be replaced by a less cumbrous appliance in the form of braces, which will allow more freedom of motion and at the same time will protect the cord and firmly hold the spinal column until complete ankylosis of the diseased vertebræ makes support no longer necessary. This part of the treatment should, if possible, be directed by an orthopedist. During all the time the above mechanical treatment is being

carried out the patient should be treated constitutionally as directed in the chapter on Tuberculosis. Without good food and fresh air a cure cannot be effected in these cases.

*Syphilitic myelitis* is to be treated with mercury and the iodids as directed in the chapter on Syphilis.

In *acute infective myelitis* the patient is to be put to bed as above directed. Ice may be applied intermittently to the spine during the first four or five days if it does not irritate the patient. As soon as the diagnosis is made, 20 c. c. of antistreptococcic serum should be administered, and this dose is to be repeated every six to twelve hours until six doses have been given. Inunctions of unguentum Credé, 2 drachms every twelve hours, should also be used over the same period of time. This treatment may be of service in modifying the inflammation if the infection be streptococcic; in every instance where the myelitis follows one of the acute infections or appears without apparent cause it should be used.

*Residual paralysis and contractures* are to be treated as recommended under Cerebral Palsies.

In those cases that are fortunate enough to make a favorable convalescence great care should be exercised in guarding them against the too early use of the muscles weakened by paralysis. These muscles require a long period of time to recover their normal strength and tone, and satisfactory convalescence may be interfered with by subjecting them to fatigue during this period.

## HEREDITARY ATAXIA

(*Friedreich's Disease*)

Hereditary ataxia is an hereditary disease of the spinal cord characterized by degeneration and sclerosis of the long posterior tracts, the cells of Clark and those of Gowers and their axons, and the spinocerebellar tracts. This is associated with a lack of development of other portions of the cord and of atrophy of its posterior roots. In addition the process frequently involves the lateral (pyramidal) tracts.

**Etiology.**—Heredity is the most important etiological factor. It occurs as a family disease, extending through a number of generations. It is a disease of childhood, beginning, as a rule, before the tenth year of life. It may, however, occur in early infancy and in adult life.

**Symptomatology.**—Ataxia is its characteristic symptom, and in many respects the symptoms resemble those of locomotor ataxia in the adult. In the beginning the child walks with his legs apart in an awkward, unsteady manner. The leg is lifted carefully and brought down suddenly as in locomotor ataxia. Romberg's sign is often present; that is to say, when standing still with the feet close together there is a swaying, uncertain movement of the body, and, if the eyes are closed, the patient falls to the floor. There may also be vibratory movements (chorciform) of the head and eyes. Later the ataxic movements become well marked in the

arms; these awkward, jerky, sudden movements of the arm may be brought about by asking the patient to pick up some object. Later on there is more or less rigidity of the muscles of the arms and legs which greatly interferes with the functions of these parts. Muscular power is gradually lost, so that late in the disease almost complete paralysis may result. The complete loss of the knee-jerk and other deep reflexes is an important symptom. Speech is slow, measured and difficult. In these cases the instep is highly arched, the toes hyperextended. Late in the disease the mind of the patient may be dulled and his expression stupid, though often the mental state remains unimpaired.

Reflex pupillary rigidity (Argyll-Robertson phenomenon) and ocular muscle palsies do not belong to the picture of Friedreich's disease as they do to tabes.

**Prognosis.**—This disease is invariably progressive. The patient in a few years becomes a hopeless cripple and perhaps an imbecile, unable to help himself in any way. These unfortunates often live far into adult life.

**Treatment.**—There is absolutely no treatment that favorably influences the course of this disease. It becomes the duty, however, of the physician to prolong the lives and minister to the comfort of these unfortunate patients. In seeking to accomplish these ends the diet may be carefully supervised, that it may serve nutritional purposes and come within the range of the child's digestive capacity. Outdoor life, wholesome, hygienic surroundings, and careful attention to the gastrointestinal canal are necessary. Orthopedic apparatus carefully designed to support weakened joints and prevent contractures may add to the comfort of the patient.

## SPINA BIFIDA

This, the most common malformation of the central nervous system in infancy, is due to defective development of the vertebral canal. It usually consists in an absence of the spinous processes of one or more vertebræ; the laminæ may also be absent. These defects open up the spinal canal, and through this opening its contents protrude, producing hernia of the spinal cord or its membranes, which is present at birth. This protrusion commonly occurs posteriorly in the median line and is usually located in the lumbar or sacral regions. In rare instances, however, the hernia may escape through a defect in the anterior portion of the spinal canal, producing a tumor, which protrudes into the lower abdominal or pelvic cavities. This latter condition is known as spina bifida anterior, or occulta. The following varieties of spina bifida are recognized: meningocele, meningomyelocele, and syringomyelocele. They are commonly associated with other congenital deformities.

**Meningocele.**—Meningocele is the rarest and simplest form of spina bifida. It is a simple hernia of the membranes of the cord, which are pushed through the opening in the spinal canal by the fluid in the arach-

noid cavity or subarachnoid space. The spinal marrow remains in position and the hernial tumor consists of spinal fluid, held by the globular dilatation of the skin and arachnoid. The dura mater opens posteriorly and becomes merged in the walls of the sac. The tumor is commonly located in the lower lumbar or sacral region, and may vary in diameter from 2 to 6 inches. It is pedunculated, translucent and not associated with paralysis or any disturbance of the functions of the cord or nerves. The spinal defect in meningocele is commonly smaller than in the more serious

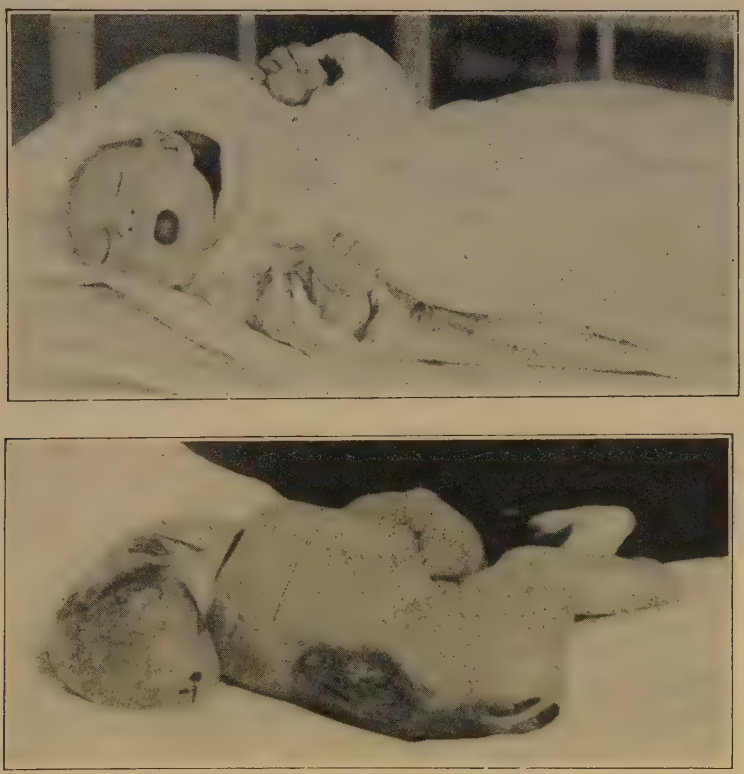


FIG. 101.—HYDRENCEPHALOCELE AND SPINA BIFIDA IN AN INFANT ONE DAY OLD.

forms of spina bifida; an X-ray picture, therefore, showing but a slight opening in the spinal canal, would indicate, in connection with the above symptom group, the presence of this form of spina bifida.

**Meningomyelocele.**—This is by far the most common and most serious form of spina bifida. In this condition the accumulation of fluid in the anterior subarachnoid space pushes the spinal marrow and its posterior membranes backward through the opening in the spinal canal, producing a true hernia of the cord and its membranes. A cystic tumor is thus formed, containing the disintegrated and attenuated fibers of the spinal cord, which have been torn by the pressure of the fluid which carried it

into the sac. Remnants of the cord are blended with the inner layer of the sac, and, in some instances, so attached to its central wall as to produce a restraining band, giving the tumor a slightly grooved or indented appearance, which is characteristic of this form of spina bifida. The tumor is located in the lower lumbar or upper sacral regions; it may vary from 1 to 4 inches in diameter; it is translucent, but not, as a rule, pedunculated. The most characteristic symptom of this form, however, is the paralysis, spastic or flaccid, of the lower extremities commonly associated with anesthesia and disturbances of the functions of the bladder or rectum. The skin covering the tumor may remain normal, but, more commonly, after a time, it becomes dark-red in color. In some instances the skin covering is absent; in others it disappears under erosion from inflammatory processes; in these cases the thin wall of the sac may rupture, leading to infection of the cord and its membranes, which soon results in the death of the patient.

**Syringomyelocele.**—Syringomyelocele is a rare form of spinal hernia produced by an increased pressure of fluid in the central canal of the cord. This pressure results in pushing the posterior half of the cord and its covering membranes through the congenital opening in the spinal canal, producing a tumor so resembling meningomyelocele that it cannot with certainty be differentiated from it. In typical cases, however, of syringomyelocele the paralysis of the lower extremities is sensory, the motor nerves not being markedly involved. It is commonly associated with hydrocephalus, the dilated ventricle of the brain being in direct communication with the tumor through the central canal of the cord.

**Diagnosis.**—While the differential diagnosis of meningomyelocele and of syringomyelocele is unimportant, it is of the greatest importance that these two conditions should be differentiated from the much rarer form of simple meningocele. The absence of paralysis and of other symptoms pointing to disturbance of the functions of the cord, with an X-ray picture showing but a small defect in the spinal column, would justify a diagnosis of meningocele and exclude the graver forms of spina bifida.

**Prognosis.**—The prognosis in simple meningocele is favorable, but in the other forms in which the spinal marrow is involved the prognosis is very unfavorable. In many of these cases the sac ruptures during labor, or shortly after birth, and the septic infection of the cord and its membranes, which quickly supervenes, produces death.

**Treatment.**—The treatment consists in protecting the tumor from friction and other injury; this is a matter of no little difficulty. Bandages, holding soft compresses covering the tumor, may be worn. In selected cases the most satisfactory treatment is the removal of the tumor mass by a surgical operation. All cases of simple meningocele, and these are rare, should be referred to the surgeon for operation. All cases associated with marked paralysis or hydrocephalus are hopeless and cannot be benefited by surgical procedures. Operation, however, is to be recommended in all infants suffering from spina bifida who have lived to be 6 or 7 months of

age, who have gained in nutrition, and who, during this time, have shown satisfactory evidence of increasing mental development and who have either no paralysis or but slight paralysis of the lower extremities. Under no conditions should the operation for spina bifida be performed until the infant has shown satisfactory evidence of both mental and physical development, and this practically precludes operation until the child is 6 or 7 months old.

## CHAPTER LXXXI

### DISEASES OF PERIPHERAL NERVES

### MULTIPLE NEURITIS

Multiple neuritis is an inflammation of peripheral nerves, which results in more or less complete loss of function to the nerves involved. It is usually symmetrical in its distribution.

**Etiology.**—Diphtheria toxins are the most common cause of multiple neuritis in children. In the report of the collective investigation by the American Pediatric Society, paralysis occurred in 9.7 per cent. of 3,384 cases of this disease which had been treated by antitoxin. It appears, therefore, that the antitoxin treatment of diphtheria, unless administered early, has little influence in preventing the subsequent development of neuritis. Influenza and malaria are perhaps the next most common causes of this disease in children, and, in rare instances, it may follow any of the acute infections. Alcohol and the metallic poisons, such as arsenic, lead, mercury and zinc, which so frequently produce this disease in the adult, are occasionally found as exciting factors in the child. Of this group of poisons Putnam finds that arsenic is the most common cause of neuritis in children.

**Pathology.**—Neuritis may be either parenchymatous or interstitial. The parenchymatous form, which occurs in diphtheria, is the one usually seen in children. In this condition the evidences of the acute inflammation of the nerve are absent, and in its stead there is a degenerative process which slowly destroys the axis cylinder and its myelin sheath; the neurilemma, or the sheath of Schwann, however, is left intact, and from the cells of this sheath the nerve may be regenerated and its function restored. In the interstitial form of this disease, which may be caused by the other acute infections, or by alcohol and the metallic poisons (lead excepted), there is also a degenerative process which may destroy the axis cylinder and the myelin sheath, but it is accompanied by an acute inflammation of the nerve, causing swelling, redness, tenderness, acute hyperemia, infiltration with round or oval cells, and proliferation of connective tissue. The acute inflammatory process in the nerve, however, subsides early and the degenerative process proceeds to the more or less complete destruction of the nerve. In this form of neuritis regeneration and restoration of func-

tion of the nerve may also occur. But the regenerative process is slow, lasting over one or two months.

**Symptomatology.**—The onset may be marked by fever, pain and general nervous irritability, but, as a rule, the paralysis develops insidiously, unannounced by acute symptoms.

In diphtheritic neuritis the paralysis nearly always begins in the soft palate and pharynx. Attention is called to this fact by difficulty in swallowing, by the nasal twang of the voice and by regurgitation of fluid foods through the nose. Examination will show that the soft palate hangs down and does not rise in phonation. Usually there are anesthesia of the mucous membrane and an absence of the palatal reflex. The involvement, generally bilateral, is occasionally one-sided. Frequently there is involvement of the ocular muscles with palsies, and especially the ciliary muscle with loss of accommodation. Involvement of the adducens betrays itself by squint and the inability to turn the eye outward; occasionally the vocal cords are paralyzed, resulting in hoarseness and aphonia. When these symptoms occur the knee-jerk should be carefully examined, as a diminution in this reflex indicates the extension of the paralysis to the lower extremities. The subsequent course of this paralysis is similar to that of multiple neuritis produced by other causes.

The other forms commonly develop following one of the other acute infections, or perhaps without apparent cause. In these cases, without preliminary throat paralysis, the child commences to have an unsteady or ataxic gait, and fails to use its feet and hands in a proper manner; during this stage there may be muscular tremor and incoördination. The paralysis first becomes noticeable in the parts of the body most remote from the central nervous system; "wrist-drop" and "foot-drop" caused by paralysis of the extensors of the wrist and foot are early and characteristic symptoms. The paralysis may then gradually extend up the arms and legs; in severe cases it involves the muscles of the trunk and neck and produces complete general paralysis, the patient being unable to make a voluntary movement. He lies limp and helpless, and when the body is lifted the head falls about from lack of muscular support. Such widespread paralysis should always suggest multiple neuritis. The paralysis, however, is not, as a rule, so widely distributed, but, whatever may be the extent, it is symmetrical, and is associated with more or less sensory paralysis. The sensory paralysis, however, is not, as a rule, complete, but partial anesthesia is common, especially during the early stages of the disease. This complete, symmetrical motor paralysis, associated with disturbances of sensation, is the characteristic paralysis of this disease. In some cases, however, the sensory disturbances are not well marked, and, as a rule, they disappear long before the motor paralysis. Pain and tenderness along the course of the affected nerves are characteristic symptoms in nearly all forms of neuritis, except that produced by diphtheria; in this form they are absent.

The nutritive function of the paralyzed nerve is also interfered with;

this results in a mild form of muscular atrophy, nothing like so marked as that which occurs in anterior poliomyelitis. The reaction of degeneration is present in the atrophied muscles. The reaction to the galvanic current is slow and feeble, and the anodal closure contraction is greater than the cathodal closure contraction. In severe cases the muscle fails to respond to any form of electrical stimulation; in these cases the atrophy is more marked. The knee-jerk is commonly absent, and other reflexes of the paralyzed part are diminished or lost.

Cardiac paralysis from involvement of the vagus and respiratory paralysis from involvement of the phrenic and intercostal nerves may occur, but they are rarely seen except in the diphtheritic form of this disease. In cardiac paralysis there may be few or no warning symptoms and death may occur quite unexpectedly. In other cases the condition of the heart is made evident by an irregular, intermittent, weak pulse, and this may be associated with coldness of the extremities and precordial distress. In respiratory paralysis there is more or less disturbance of the respiratory rhythm with cyanosis and dyspnea, and as the diaphragm is commonly paralyzed, abdominal respiratory movements are absent; these symptoms are associated with great anxiety on the part of the patient.

**Diagnosis.**—The differential diagnosis of multiple neuritis from other forms of paralysis in childhood is outlined under Anterior Poliomyelitis.

**Course.**—The paralysis usually increases in severity or remains stationary for four or five weeks. Improvement then gradually sets in, from two to four months being required for the complete restoration of function. The great majority of these cases end in complete recovery; in some instances, however, a residual paralysis due to destruction of nerves and atrophy of muscles may occur. A fatal termination from cardiac and respiratory paralysis is rare, except in the diphtheritic form of this disease.

**Treatment.**—Absolute rest in bed and freedom from conditions that produce nervous irritation should be insisted upon. Calomel followed by Rochelle salts or castor oil should begin the treatment. The dietetic treatment of these cases is important, since a disturbed gastrointestinal canal may greatly interfere with nutrition and unfavorably influence the progress of the disease. A careful search in every case should be made for the cause of the disease. If metallic poisoning should be found, its source should be removed and eliminative treatment instituted. If malaria or syphilis be suspected, the specific treatment for these diseases should be given. In the great majority of cases the treatment should be mildly eliminative and otherwise symptomatic. The eliminative treatment consists in mild, warm alkaline baths, in the free use of water, and in keeping the excretory organs in good condition. One or two warm baths each day will not only promote elimination through the skin, but will greatly modify the pain and discomfort from which these patients suffer. Drinking large quantities of water should also be insisted upon, as this is one of the most important therapeutic measures. Warm applications made with flannels or hop bags may modify the pain and tenderness along the nerves.

Phenacetin may be used to relieve pain, the bromids to overcome the general nervous irritability, and veronal to produce sleep. Opium is rarely, if ever, necessary, and should be avoided except in those cases where the pain is great and does not yield to simpler measures. The objection to opium is that it constipates and interferes with elimination and nutrition. Electricity and general massage are of value late in the disease in keeping up the tone and nutrition of muscles and in bringing about an earlier restoration of function in the paralyzed parts. They are to be used as directed under Anterior Poliomyelitis. Strychnin, iron, cod-liver oil and other tonic treatment may be employed during the long period in which the restoration of function to the paralyzed muscle is being brought about. If cardiac and respiratory paralysis threaten, the patient should be kept absolutely quiet and not allowed to do for himself anything that can be done by others, and in the event that these dangerous symptoms commence to subside, the same absolute quiet should be insisted upon for a week or more after all symptoms of this character have disappeared. In such cases a fatal termination may sometimes be precipitated by rising up in bed. In cardiac failure strychnin and strophanthus are indicated; in respiratory failure strychnin is perhaps the best remedy; these drugs should be given hypodermically.

## FACIAL PARALYSIS

(*Bell's Palsy*)

**Etiology.**—Facial paralysis is due to a paralysis of the seventh nerve. It may occur in the new-born from injury to this nerve by obstetrical forceps or from pressure of the face against the pelvic bones during protracted labor.

In older children facial paralysis may be due to a peripheral neuritis of this nerve resulting from "cold." These cases are not fully understood and are usually spoken of as rheumatic, although there may be no other rheumatic symptoms present. This group includes all the idiopathic cases for which a definite exciting factor cannot be found; many of them are perhaps toxic in origin. Another group of cases are due to injury of the nerve from disease of the petrous portion of the temporal bone produced by chronic otitis media or to injury from mastoid and other ear and face operations, or to parotitis and other inflammatory and traumatic conditions involving the tissues about the lobe of the ear.

Intracranial lesions, such as tumors of the brain, basilar meningitis and fracture of the skull, may produce this same form of palsy without involving other nerves.

Excluding the birth palsies the disease is very rare during infancy. In early childhood the ear cases are most commonly seen, and after the seventh year the idiopathic cases are most frequent.

**Symptomatology.**—The palsy is purely a motor one; the sensory nerves are not involved; there are no pain or constitutional symptoms. The par-

alysis of the face is the only symptom, except in those cases that are due to internal ear or intracranial disease. In these cases the symptoms of the causative condition were present before the paralysis and continue after its development. There is a complete motor paralysis of the muscles of one side of the face, which produces a characteristic symptom group; the eye, as Bell noted, cannot be closed, efforts to accomplish this being associated with an upward movement of the eyeball; the face on the affected side is expressionless and attempts to move it produce grotesque expressions. There may be difficulty in talking, the child mouthing its words. Whistling, blowing, laughing, or opening the mouth develops a marked asymmetry in the two sides of the face; on the healthy side the angle of the mouth is drawn upward and the deep nasolabial fold is in contrast to the smooth face on the opposite side. In lifting the eyebrows, the forehead on the paralyzed side remains smooth in contrast with the wrinkling on the opposite side, and in attempts at showing the teeth the mouth assumes an irregular shape, the line between the upper and lower incisors, instead of being continuous, shows deviation.

In those cases where the nerve is permanently injured atrophy of the muscles occurs. This leads to a wasting of one side of the face and to the development of the reaction of degeneration in the paralyzed muscles.

**Diagnosis.**—From other forms of paralysis facial palsy can easily be differentiated by remembering that it is a motor nerve paralysis confined to the muscles of the face. There should be little difficulty in determining the cause of the facial palsy. Intracranial lesions, such as meningitis and brain tumors, announce their existence by characteristic symptoms. If facial paralysis be associated with deafness without apparent disease of the ear and weakness of the outward rotator of the eye, other symptoms of cerebellar tumor should be sought for. If a chronic otitis media exists it is probably the cause of the disease. In the absence of other causative factors it is assumed that the condition is due to cold or rheumatism (idiopathic).

**Prognosis.**—The prognosis will depend largely upon the causative condition. In birth palsies and in the idiopathic cases, due to "cold," the prognosis is generally good; complete recovery occurs in from one to six months. The electrical reactions in these cases will materially assist in determining the course of the disease; if the muscles react to both the faradic and galvanic currents in a normal manner at the end of the first week, recovery will be rapid, but if the muscles at this time fail to respond to the faradic current, but yet respond to the galvanic current, recovery may not be expected in less than two or three months. In the more severe cases, where the muscle not only fails to respond to the faradic current, but shows the reaction of degeneration to galvanism, and contracts but feebly and slowly to strong currents, the paralysis may continue for a year, and in some cases may be permanent. The prognosis in those cases associated with disease of the ear will depend altogether upon the character of the lesion. If the nerve be cut or otherwise destroyed, the paralysis

may be permanent, but, in the majority of cases, the injury to the nerve is of such a character that the removal of the exciting cause results in a slow but complete recovery.

**Treatment.**—Cases due to disease of the ear or temporal bone require proper surgical treatment. Those occurring without apparent cause may be given salicylate of soda, as recommended in the chapter on Rheumatism; this treatment is especially indicated if the facial paralysis is associated with sore throat or pharyngitis. The salicylate treatment, however, should not be continued longer than three or four days, and is contraindicated if disease of the ear be present. Following this treatment the patient's general health should be looked after by proper food, outdoor life and tonics. Iron, cod-liver oil and the malt preparations may be indicated. The most important part of the treatment, however, consists in keeping up the nutrition of the paralyzed muscles, and this should be done by massage and electricity, as directed under Anterior Poliomyelitis, but this treatment should not be begun until after the second or third week. In the use of galvanism, mild currents just strong enough to produce muscular contractions should be used.

Blisters and other forms of counter-irritation may be applied beneath the lobe of the ear; this is sometimes of value. In properly selected cases nerve transplantation may be of benefit. In this operation the facial nerve is cut and transplanted in the sheath of the hypoglossal nerve. Some good results have been reported from this operation. This surgical measure is indicated only in those cases where the electrical reactions justify an unfavorable prognosis, or where the nerve has been cut in surgical operations.

## PROGRESSIVE MUSCULAR DYSTROPHY

This term is used to embrace a group of syndromes characterized by more or less widespread atrophy and loss of function of the voluntary muscles. The atrophy is associated with fatty degeneration of muscle fibers and proliferation of connective tissue. In the pseudohypertrophic form the apparent increase in size of certain muscles is due to fatty deposits; a true hypertrophy, however, of certain muscle fibers also occurs. In the peroneal type of this disease (if for convenience one may include it under this heading) the peripheral nerve changes discovered by Hoffman are believed to be the cause of muscular atrophy. In the other types no changes either in the peripheral or central nervous system have been found.

**Etiology.**—The causes of this disease are unknown. It is believed to be due largely to hereditary defects; at any rate it is distinctly hereditary, and, although boys are more commonly affected than girls, the hereditary transmission occurs almost always through the mother; a number of cases may occur in the same family.

**Symptomatology.**—Pseudohypertrophic Form.—This is the most common of the clinical types of this affection. It develops, as do all the

others, slowly and insidiously, unmarked by acute constitutional symptoms. It begins, as a rule, between the second and seventh years of life, is characterized by progressive loss of strength in the voluntary muscles, the patient becoming more and more helpless until he is hopelessly bedridden. But as the involuntary muscles are not involved and the vital organs are not affected, the patient may live many years, to die from some intercurrent disease.

The first symptom noticed is a clumsiness of gait associated with an increase in the size of the calf of the leg. There is a notable loss of power and endurance in the muscles of the lower extremities. As the disease progresses the hypertrophy of the calves is in contrast with the atrophy of the muscles of the thigh, back, shoulder, chest and upper arm. With the increasing atrophy there are gradual loss of power and final loss of func-

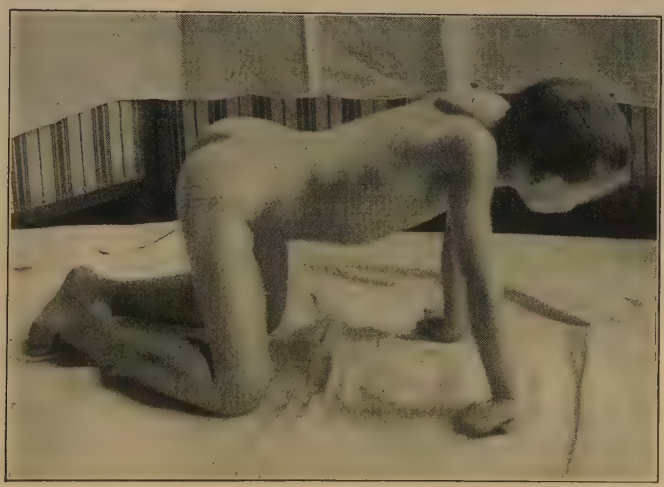


FIG. 102.—PROGRESSIVE MUSCULAR DYSTROPHY, PSEUDOHYPERTROPHIC FORM.

tion on the part of the muscles. In the earlier stages, before the arm muscles are noticeably involved, the patient uses his hands to assist in making movements that are ordinarily accomplished by the now weakened muscles of the leg and back, and in doing so he assumes positions which are very characteristic of this disease. Some of these positions are illustrated in the following series of photographs which represent different positions assumed by the child in lifting himself from the floor. If placed on his back the patient slowly turns on his face, lifts himself upon his arms, and then, by the aid of his hands, "he climbs up himself" until he finally reaches the upright position, and then, with legs widely separated, he walks with a clumsy waddling gait. The forward curvature of the spine, which increases with the wasting of the deep muscles of the back, is shown in the accompanying figures. The reflexes are diminished in the atrophied parts and the electrical reactions are feeble. As the disease progresses the patient finally becomes a helpless, bedridden invalid. Nystag-

mus, difficulty in speech, and lack of mental development may be present in these cases.

Erb's Juvenile Type.—This form usually occurs between the tenth and sixteenth year, and begins in the muscles surrounding the shoulder. They gradually lose their power, and may be wasted or hypertrophied. With the loss of function in these muscles the patient is unable to lift his arms, the atrophy extends to other muscles of the upper arm, back, thighs, and legs. With the atrophy and progressive loss of function of these muscles the patient gradually loses the power of locomotion, and becomes as helpless as in the pseudohypertrophic type.

Landouzy-Dejerine Type.—This type, like the pseudohypertrophic

Fig. 103.—Same as Fig. 102.

type, begins in early life, but is differentiated from the others by the fact that the muscular atrophy begins in the face. It is first noticed about the mouth; the lips are thickened and everted, the mouth is slightly open, the patient being unable to close it. The muscles of the lower part of the face, neck and shoulder girdle, gradually become involved with a progressive muscular atrophy and loss of function. The subsequent history of these cases is similar to those of the Erb type.

Peroneal Type.—This type of muscular atrophy is usually classed as a separate disease, since it is believed to be due to degenerative changes in the peripheral nerves. The atrophy begins in the muscles of the feet and spreads to the muscles of the calf, producing a general atrophy, and in time, complete loss of power of the muscles below the knee; the disease,

as a rule, is confined to this part of the body, but it may spread to the thigh, hand, forearm and shoulder.

Sensory changes in the atrophied part may be present, but total loss of sensation is uncommon. The reflexes below the knee and in other atrophied portions of the body are diminished or lost. Although these patients are hopelessly crippled, they may live for a long time. In those cases where the disease is limited to the legs below the knees the prognosis, so far as life is concerned, is especially good; in the other cases, however, where the disease extends to the thighs, arms, shoulders and other voluntary muscles, the patients become hopelessly bedridden, and usually die from some intercurrent disease.

Fig. 104.—Same as Fig. 102.

**Treatment.**—All of the above-named types are progressive and run their course uninfluenced by any kind of treatment. All the physician can do, therefore, is to treat them symptomatically and look to their general health. They should be carefully fed, live an outdoor life, and be placed under the best hygienic conditions. As long as they are able to use their muscles they should be allowed to do so. In accomplishing this end orthopedic appliances to overcome contractures and to support the spine, the ankles, and the knees may be of great service in keeping the patient on his feet for a longer time than would otherwise be possible.

Massage, passive movements and electricity may be used to promote circulation and stimulate the nutrition of the slowly degenerating muscles.

## CHAPTER LXXXII

### GENERAL NERVOUS DISEASES

### DISORDERS OF SLEEP

Sleep is the physiological rest which the tired organism demands to repair the fatigue changes incident to the physiological activity of cells, especially those of the nervous and muscular systems. The physiological activity of all the organs of the body alternates with periods of relative repose. This repose is absolutely necessary to the vital activity of cells.

In the higher animals the central nervous system rests at least once in twenty-four hours, and this condition of rest is called sleep. Normal sleep is characterized by loss of consciousness, loss of voluntary inhibitory control of motor and mental acts, and more or less complete loss of all the special senses. Sight goes first, probably taste and smell next, and finally touch and hearing disappear as sleep becomes profound. During sleep all of the higher functions of the brain are held more or less in abeyance, and the involuntary inhibitory control of motor and mental acts is also partially lost. The discharge of nervous stimuli to all the organs of the body is greatly diminished, and, as a result, there are more or less relaxation of the muscular system and a feebler functional activity of nearly all the important glands.

During sleep, however, the capacity of the central nervous system to react to peripheral stimuli is not altogether lost. But the more profound the sleep the stronger must the peripheral stimulation be to make any impression upon the nerve centers. In the very beginning of sleep the nervous system may respond very actively to slight external stimuli, producing muscular twitchings of the body, which may be severe enough to arouse the individual with the knowledge that this spasmodic contraction has occurred. These phenomena, however, are more likely to occur in highly nervous individuals, the nervousness being produced by unusual activity of the brain before going to bed, or by an excitable condition of the higher nerve centers produced by toxins. While this condition of increased reflex excitability at the beginning of sleep can scarcely be said to be physiological, yet it is made possible by the fact that the higher nerve centers, which exercise inhibitory control over the lower, are the first to lose their functions under the influence of sleep; and, as sleep becomes more and more profound, the entire nervous system gradually sinks into a condition of more or less complete repose, the motor centers at the base of the brain, and the reflex centers of the cord being the last to come under its sedative influence. When the entire nervous system has come under the influence of profound sleep, the reflex centers of the brain and cord are not so readily excited to action by peripheral stimuli as they are in the beginning of sleep, when the inhibitory centers are in repose, and the motor centers have not yet lost their normal excitability. During the first hour sleep becomes more and more profound. At the end of this time the higher nerve centers are very profoundly under its influence, and it requires comparatively powerful stimuli to bring the individual back to consciousness. During the second hour sleep becomes gradually less profound, and from this time on a comparatively slight stimulus is sufficient to awaken the individual. The profound sleep of the first two hours has been likened to a condition of narcotism, which slowly passes off, leaving the individual still unconscious, but easily aroused. The lower motor centers of the brain and spinal cord maintain about the same degree of irritability from the beginning to the close of sleep. They are apparently not influenced, as the higher centers are, by the narcotism of the first and second hours of sleep.

The healthy newly born infant sleeps nearly all of the time, at least twenty out of the twenty-four hours. During the first month the normal infant is awake about four hours in the twenty-four. From this time on the child requires slightly less sleep, so that at six or eight months he is sleeping sixteen hours in the twenty-four, and at the age of one year he sleeps from twelve to fourteen hours. During the first few days of life sleep is heavy, owing to the fact that the organs for receiving and carrying peripheral stimuli to the central nervous system are not yet fully developed. From this time on during the next month sleep becomes less profound, and from the end of the third month to the end of the second year sleep is not so deep as it is after the third or fourth year, when the heavy sleep of childhood is seen. It is at this time in the life of the individual that the profound narcotism of the early hours of sleep is most noticeable.

The most common disorders of sleep are night-terrors, somnambulism, and insomnia. Of these the most important is night-terrors, or pavor nocturnus.

## PAVOR NOCTURNUS

Pavor nocturnus is a neurosis dependent upon an abnormally irritable nervous system, easily excited by reflex stimuli having their origin in distant parts of the body, or in the cortical centers themselves. It is characterized by a night-terror which finds expression in the child's screaming or crying out in a panic of fright during sleep.

**Etiology.**—Predisposing Causes.—Heredity is a very potent etiological factor. In the most severe cases there is commonly a well-marked neurotic family history, and such neuroses as epilepsy, hysteria, chorea, migraine and neurasthenia not uncommonly occur in the family histories. This strong hereditary taint predisposes these children to muscular twitchings, convulsions and reflex neuroses of all kinds. The particular defect of the nervous system which is inherited is a feeble inhibitory control of mental processes and motor acts. This may explain the relationship existing between epilepsy, infantile eclampsia, and night-terrors, which appear to be present in some families. Beyond this there is perhaps no direct connection between these neuroses. While a neurotic family history, resulting in an extremely irritable nervous system under feeble inhibitory control, is present in many of the more severe cases of night-terrors, this factor is by no means so well marked in the milder types of this disorder. In some instances the excitable nervous system seems to be wholly dependent upon other factors entirely foreign to hereditary influences.

Malnutrition is an important factor in developing irritability of the nervous system in young children, and the common causes of malnutrition, such as lymph-node tuberculosis, chronic diseases of the gastrointestinal tract, chronic malaria, hereditary syphilis, and rachitis, with improper food, impure air, and bad hygiene, may therefore be important predisposing factors of night-terrors.

Mental overwork and excitement, when coupled with physical in-

feriority, are most potent factors in producing the highly irritable state of the nervous system which makes possible the development of this syndrome. School life, with its mental grind, persistent excitation, close confinement, and eye-strain, may be a factor in the development of night-terrors.

Exciting Causes.—The normal irritability of the nervous system of the child, having been exaggerated by bad heredity, malnutrition, mental overwork, or nervous excitation, makes it possible for certain reflex exciting causes to develop an attack of night-terrors. The intestinal canal is one of the most important sources of this reflex irritation; undigested food, improper food, excess of food, intestinal worms, and intestinal fermentations, with the intestinal toxins which they produce, may all act either directly or indirectly as exciting factors. Adenoids, enlarged tonsils, and nasal obstructions that interfere with normal breathing during sleep, may either act as reflex factors or they may act by producing a partial asphyxia, and thus excite an attack of night-terrors.

In many cases, however, the reflex factors are absent, or perhaps it might be better to say are so slight that they cannot be readily discovered. In these cases the attack is apparently excited by a horrible dream, which has its origin either in some alarming occurrence of the previous day or in the overstimulation of the emotional centers by blood-curdling tales or exciting fairy stories. The nervous systems of extremely neurotic children may be so excited by punishment, by fits of anger, and by fright that they fall asleep with the incidents of the day still impressed upon their nervous systems, and, as a result, the cortical centers do not come profoundly under the reposeful influences of sleep, and in the paroxysm of night-terrors which supervenes, the horrible vision which presents itself to the child in his night-terror is but an exaggerated reflex of some mental impression which he received during the day.

**Symptomatology.**—Silbermann divided night-terrors into two rather distinct clinical types, which for the most part have been recognized by recent writers. One of these he called Idiopathic Night-Terrors, and the other Symptomatic Night-Terrors. The idiopathic type is of central or cortical origin, and the symptomatic of peripheral origin. In the description which follows, these two types will be recognized.

### NIGHT-TERRORS

**Central or idiopathic night-terrors** has for its most important etiological factor an extremely excitable nervous system under feeble inhibitory control, which has been inherited from neurotic parents. In the family history of these cases, hysteria, neurasthenia, and the convulsive neuroses, all of which are largely dependent upon feeble inhibition, are common. The inherited neurotic condition may also be aggravated by malnutrition and improper training. There can be little doubt, however, that even in these cases peripheral irritation plays a part in touching off the paroxysm;

but the central nervous system is in such a state of excitability, and under such feeble inhibitory control, that a slight peripheral irritation produces a maximum result, and for these reasons it is commonly disregarded or overlooked. Idiopathic night-terrors occur in the great majority of instances between the ages of two and eight years. This is the period of life when feeble inhibitory control of cortical and other centers is responsible for many of the graver nervous diseases, such as eclampsia, epilepsy and chorea.

The Paroxysm.—A neurotic child, with his nervous system unusually excited by the incidents of the day, falls asleep, and after an hour or two suddenly starts in his sleep with a cry of terror that alarms the household. A moment later he is found apparently wide awake, sitting up in bed, or crouching on the floor in a state of wild excitement, staring and pointing at some horrible, imaginary object which he seems to see with great distinctness. He trembles with fear and gesticulates wildly, calling for assistance, but when spoken to fails to recognize his nurse, who is vainly endeavoring to arouse him to consciousness. He may call out the name of some man or animal who he thinks is about to do him injury. After a few minutes of this agonizing fear the attack spends its force, the excitement gradually passes away, and the little patient falls back upon the pillow and becomes quiet in sleep, which may continue without further disturbance until morning. In many instances the child will go through an attack of this kind without recovering consciousness; in other words, the whole attack occurs during sleep. In other instances the strenuous efforts of the attendants may arouse the child to a vague consciousness, or, rather, semiconsciousness, during which, in a dazed way, he recognizes his surroundings, and then quickly drops asleep, and the next morning has little or no recollection of what has occurred during the night. According to Silbermann, Coutts, and other observers, the seeing of visions is the most characteristic feature of these attacks of central or idiopathic night-terrors. Similar attacks may occur for a number of nights in succession, or there may be an interval of weeks or months between them, but they always present very much the same clinical picture, although they vary in intensity.

Incontinence of urine may occur or the child may, at the close of the attack, make known his wants, and after seeking the commode pass urine or have a movement from the bowels, as though he were entirely conscious of his actions, and yet give no other evidence of being conscious of his surroundings. He returns to bed, continues his sleep, and the next morning has no recollection of these occurrences.

The central type of night-terrors is believed by many writers to be closely related to epilepsy, and quite a number of cases of epilepsy have been reported in which night-terrors occurred as a part of their early history. Concerning this relationship, however, I am quite in accord with the opinion expressed by Charles Putnam in his excellent paper on this subject in the "Cyclopedia of the Diseases of Children." He says: "Al-

together, the connection between night-terrors and epilepsy, in so far as they are separate diseases, is no clearer than that between any two of the neuroses, and yet, inasmuch as attacks closely resembling night-terrors are occasionally only symptoms of epilepsy, it is well to watch carefully for a time before deciding that epilepsy is not present."

**Symptomatic night-terrors** are more common in childhood, but may occur at any age, and are much more frequent than idiopathic night-terrors. In symptomatic or peripheral night-terrors the essential ETIOLOGICAL FACTOR is outside the nervous system in some peripheral excitation. Children suffering from this symptom-complex have, as a rule, unstable and irritable nervous systems, but this nervous instability, instead of being hereditary, is usually acquired. Chronic malnutrition and other factors capable of producing an unstable nervous system in an otherwise healthy child may commonly be observed. The reflex factors above noted as having their origin in the intestinal canal, nose, throat, and other organs are present, and can usually be very readily discovered.

THE PAROXYSM.—The child falls asleep and may toss restlessly for an hour or two before the reflex irritation to the nervous centers culminates in an attack of night-terrors. The patient screams with terror, sits up in bed, or runs about the room. He is wildly excited, trembles with fear, and exhibits a very marked, but, as a rule, undefined, terror. He sees no visions and hears no noises, and responds to the efforts of his attendants to arouse him. He recognizes his attendants and seeks consolation from them. His nervous fears are soon quieted, and he falls asleep, to awaken the next morning with perhaps a vague recollection of the occurrences of the night.

**Clinical Pictures.**—Silbermann, and after him Coutts, have called attention to the differences in the clinical pictures portrayed in the two types of night-terrors. Coutts uses the term nightmare to describe the class of cases which Silbermann speaks of as symptomatic or peripheral. As Coutts puts it, the chief distinction between these two symptom groups is that the one suffering from idiopathic night-terrors "sees visions," while the one suffering from symptomatic night-terrors merely "dreams dreams." Silbermann expresses the same idea by saying that the former is characterized by objective terror and the latter by subjective terror. It may be added also that in the idiopathic form the terror is more real, the mental excitement greater, and the condition of unconsciousness more profound.

Notwithstanding the differences in the clinical pictures which the two types of night-terrors present, I am not prepared to say that they are distinct clinical entities. I am rather inclined to believe that the idiopathic type of this disorder presents the aggravated clinical picture as it may occur in highly neurotic children, whose mental and motor mechanisms are under feeble inhibitory control. Between this extreme type and the milder attacks of symptomatic night-terrors, due almost wholly to strong reflex excitation of an almost normal nervous system, there is indeed a wide difference in the clinical pictures presented, but certainly not more so than there is in epilepsy or other neuroses. In this regard I quite agree with

Putnam, who says: "It is hard to convince one's self that there are two classes so definitely separated from each other. It is true that between two individual cases there may be a vast difference in all the particulars mentioned by Silbermann, but, taking all cases together, the degrees of difference are so slight that it is almost, or quite, impossible to draw a line of demarcation."

**Prognosis.**—In the symptomatic form the prognosis is very good, because it is produced by etiological factors which can readily be removed by appropriate treatment. In the idiopathic form the prognosis is not so good, and depends largely upon the gravity of the underlying hereditary taint. All of these cases, however, should yield to appropriate treatment, but idiopathic night-terrors should call attention to, and demand treatment for, the underlying hereditary condition.

**Treatment.**—In beginning the treatment of all these cases the intestinal canal must be carefully scrutinized and all possible reflex irritation from this source removed. A preliminary cathartic followed by a carefully regulated diet with a light evening meal should be a part of the treatment in every case. It is impossible to lay too much stress upon the rôle which disorders of the gastrointestinal canal play in these cases. It is incumbent upon the physician, therefore, to thoroughly satisfy himself that the intestinal canal of the child is no longer a source of irritation or intoxication to the nervous system, and in doing this he must remember that intestinal toxemia may be present without any pronounced symptoms on the part of the gastrointestinal tract. Enlarged tonsils, adenoids, and nasal obstructions of all kinds, as well as all other discoverable causes of reflex irritation, should be removed.

The child's general health should be carefully looked after. A diet should be selected with reference to the character of the malnutrition present. Tonics such as iron, arsenic, cod-liver oil, or malt containing diastase may be indicated in individual cases. An outdoor life, with an abundance of sunshine and fresh air, is also important. In the idiopathic cases the child's nervous system should be as carefully shielded from mental strain and nervous excitement as if it were suffering from one of the graver neuroses. The medical treatment consists in giving the bromids of sodium, or potassium, in five- or ten-grain doses at bedtime. It is best to combine with this a dose of tincture of belladonna suitable to the age of the child (one to four minims). The bromid of potash, and belladonna will, as a rule, readily control the paroxysms, and, after four or five nights, all sedative medication may cease; in severe cases, however, it may be necessary to give this prescription for weeks at a time.

## INSOMNIA

Prolonged insomnia, as it occurs in the adult, lasting through the greater portion of the night, is uncommon in children; when it does occur it is a symptom of some more or less serious disease.

Disturbed or unrefreshing sleep, with possibly a few hours of wakefulness, is common in childhood, and it is this condition, rather than true insomnia, which here interests us.

**Etiology.**—Disturbed sleep is produced by very much the same etiological factors as night-terrors. A general nervous irritability is probably the most important underlying factor; it may be a matter of heredity, it may be produced by chronic malnutrition, it may occur in the convalescence from acute infections, and it may be very greatly exaggerated by more or less constant nervous excitement. The mental stimulation and strain of school life with night study, and the anxiety which sensitive children have concerning the lessons of the following day, may in older children be causes of disturbed sleep. In infancy nervous excitement is also a cause of restless sleep. The habit of constantly entertaining infants, and constantly attracting their attention, and bringing them into the whirl and excitement of the living-room, where they may be observed and commented upon, cannot be too severely condemned. Filling young minds with exciting stories before they are put to bed predisposes to dreams and disturbed sleep. Lack of proper training is, in the young infant, the most potent of all causes of insomnia. Rocking them to sleep, lifting and fondling them every time they make an outcry, and feeding them at night are causes which produce insomnia. Disturbances of digestion are important direct exciting causes. Overfeeding and improper feeding may develop in the intestinal canal important reflex and toxic factors which, by their action on the nervous system, may disturb sleep. In infants intestinal fermentation may, by the development of gases, produce colic. This may also occur in older children, but, as a rule, constipation, with more or less obscure intestinal toxemia, is with them a more important factor of nocturnal restlessness. In very young infants hunger may be a cause of sleeplessness. Poorly ventilated and overheated rooms, with lack of fresh air, heavy and uncomfortable bed-clothing, dentition, otitis, adenoids, enlarged tonsils, and nasal obstructions may cause restlessness at night.

**Varieties.**—There are two rather distinct types of insomnia. In one the child retires, and, unlike the normal child, does not fall asleep promptly. It rolls restlessly in bed, very often is tormented by frequent desire to urinate, betrays its sensory irritability in this manner and also by its extreme sensitiveness to even the slightest noises, such as the ticking of a clock, the creaking of the shutters, and the passing of the street cars. Finally, after two or three hours, it may fall asleep, but is usually a so-called "light" sleeper.

In the other type sleep comes at once, but the child awakens in the middle of the night, and thereafter is unable to secure further sleep. Here, as in the first-named type, there will often be frequent micturition, flushing of the skin, aversion to much bed-covering, frequent punctilious adjustment of the bed and pillows. Both types may exhibit a tendency to dreams, pleasant or unpleasant, as well as to recurring chains of thoughts peculiar to each individual.

It is unnecessary to state that such children do not awaken "refreshed," but are prone to manifest the symptom-complex of abnormal fatigue. That the foundation for future neurasthenia is furnished by such prolonged sleeplessness goes without saying.

**Treatment.**—The PROPHYLACTIC TREATMENT, which should begin when the child is born, is of the utmost importance. This consists in carefully regulating the life of the infant, shielding it from excitement, feeding it at regular intervals, and insisting from the beginning that the night shall be devoted to sleep. It is a comparatively simple matter to establish a routine regularity which will firmly engraft upon the infant the habit of sleeping profoundly throughout the night. This habit, when once established and closely adhered to, will do much to overcome the nervous irritability which the infant may have inherited. As the child grows older this regularity in eating and sleeping should be carefully adhered to, and the child should be given a light evening meal and put to bed soon afterward.

TREATMENT OF THE CONDITION.—The treatment consists in attempting to establish the regularity, above referred to, with which the lack of proper training has interfered. An effort should be made to discover the essential causes of the sleeplessness. Disturbances of the intestinal tract should be carefully treated, and all possible causes of reflex irritation, whether they occur in the nose, throat, or elsewhere, should be removed. The child should sleep in a well-ventilated and not overheated room, and the bedclothing should be properly adjusted to the season of the year. If it suffers from cold feet, a warm bath at night with a hot-water bottle to the feet may assist in overcoming the sleeplessness. Shower baths are of great value; in winter the hot and cold shower or spray, in summer a moderately cold shower. Overpressure at school and mental excitement of all kinds, especially just before going to bed, should be avoided.

Insomnia, occurring as an acute condition in an otherwise healthy infant, should lead one to suspect acute intestinal disturbance. Intestinal pain produced by colic may be relieved by an enema, and the child that has fretted and tossed for hours may fall asleep.

The use of medicines to promote sleep in children is rarely necessary, unless the restlessness is produced by some acute febrile condition. Bromids of potash and sodium are perhaps the most justifiable remedies under these conditions. Other hypnotics, which are so valuable in the treatment of insomnia in the adult, are of doubtful value in the child.

## SOMNAMBULISM

Somnambulism, or sleep-walking, has very much the same etiological factors as night-terrors and insomnia.

The somnambulist, soundly asleep and apparently unconscious, with his special senses in abeyance, may rise, walk, or run about in the dark, avoiding objects and performing difficult and apparently purposive acts

quite as dextrously as he could when awake, but when aroused from this state he is unconscious of what has transpired. Somnambulism is not uncommonly observed in children, but the marvelously complicated movements which have been accredited to adult sleep-walkers have not been noted in the child. Children, however, may get out of bed and walk or run about the room in the pursuit of some object, or with a definite purpose suggested by a dream, which the child is acting.

Sleep-talking may be combined with sleep-walking. I once witnessed a performance of this kind in a child seven years of age. This child during the day had been much interested in seeing his dog Towser catch and kill rats as they were one by one liberated from a trap. In the early hours of the night he sprang from bed and ran in the dark through the house, calling to his dog, "Rats, Towser, rats! Towser, here they are!" and, for some minutes, avoiding furniture and directing his movements with great accuracy, he led the chase until he was finally captured by his mother, and in his half-dazed state led back to bed and to sleep. The next morning he knew nothing of the occurrence. Rarely somnambulism may be confounded with a graver type of automatic subconscious activity, the psychic equivalent of an epileptic attack.

The treatment for this condition is the same as that above outlined for Insomnia.

## ECLAMPSIA IN INFANTS AND CHILDREN

A convulsion is a sudden discharge of motor nerve force resulting in violent and rapid muscular contractions of one or more parts of the body. It is not a distinct disease, but a symptom group which may be produced by a great variety of causes.

**Etiology.**—Predisposing Causes.—*Age.*—Infants during the first few weeks of life are comparatively immune to eclampsia, but from the fourth month to the end of the second year they are especially predisposed to all kinds of convulsive disorders. In the third year of life convulsions become less frequent, and from this time on in the normally developed child they are but slightly more common than they are in the adult. The explanation for the varying predisposition of the infant and young child to convulsive disorders at different periods of its life can be largely found in the anatomical and physiological development of the nervous system.

The excitation of cerebral motor centers cannot readily produce convulsive disorders in the very young infant, because the discharge of nerve force from these centers is not readily communicated to the spinal reflex centers, since at this early period of life the fibers of the pyramidal tracts have not fully developed their myelin sheaths, and are not, therefore, capable of readily transmitting impulses from the cortical centers to the spinal cells. The development of these myelin sheaths, however, gradually goes on, so that the pyramidal tracts have their functions sufficiently developed to place the lower spinomuscular neurons and the cerebral mo-

tor centers in close touch by the time the child is three or four months of age. In addition to this the following peculiarities of the nervous system of the young infant protect it to a certain degree from convulsive disorders: The cerebral motor areas at birth are but poorly developed and do not, therefore, readily react to excitation. These motor areas, however, are rapidly developed, so that early in the life of the infant they become very sensitive to all kinds of stimulation. The reflex centers in the spinal cord at birth are poorly developed and not easily excited, but these centers also become very excitable after the third or fourth month of life. Both the sensory and motor peripheral nerves, which are a necessary part of the reflex arc through which convulsive disorders find expression, have a very low degree of irritability in the newly-born child. A little later in the life of the infant these nerves become hypersensitive. This hyperexcitability of peripheral nerves is much more marked in some children than in others, and constitutes the spasmophilic diathesis; spasmophilia is an important predisposing cause of eclampsia, tetany, and perhaps other convulsive disorders in infancy.

The most important peculiarity of the young nervous system is the comparative lack of both automatic and voluntary inhibition, by reason of which higher nerve centers exercise little or no inhibitory control over the lower convulsive and reflex centers. *Lack of inhibition* is especially important, since it continues to a greater or less degree throughout infancy and young childhood; this explains the comparative frequency of convulsions between the fourth month and the beginning of the third year, when both sensory and motor peripheral nerves are very excitable; the cerebral motor centers are very active and easily irritated; the spinal reflex centers are easily excited, and the communications through the pyramidal tracts between the cerebral motor centers and the spinal cord cells have become very close through the development of the myelin sheaths of the fibers of the pyramidal tracts. Later in the life of the child convulsive disorders are less common, because the spasmophilia has largely disappeared and the whole nervous system is now less irritable and more stable, and the inhibitory function of the cortical over the subcortical and lower spinal neurons is more firmly established.

*Heredity* is a very important predisposing cause to convulsive disorders. Whole families of children will be found who have convulsions from very slight causes. The child may inherit, from neurotic ancestors, unstable and irritable nerve centers under feeble inhibitory control, which predispose it to all kinds of functional nervous diseases.

*Rachitis* plays such an important rôle in the etiology of infantile convulsions that it is sometimes classed as a direct, rather than an exciting, cause. Rickets predisposes to neurotic disease in general and to convulsive disorders in particular, because it is the most common and the most profound form of malnutrition which occurs in infancy; it acts by exaggerating all those physiological weaknesses of the nervous system of the infant, which may predispose it to convulsions, and as a result an infant

during the second six months of life, suffering from well-marked rickets, may have convulsions from causes so slight that they ofttimes cannot be detected. Other forms of malnutrition occurring during the convulsive age may predispose to eclampsia. Among these the following may be mentioned: chronic gastroenteritis, hereditary syphilis, tuberculosis, scurvy, and anemia.

Exciting Causes.—Intestinal toxins, usually of bacterial origin, are by far the most common exciting causes of convulsive disorders in infants under two years of age. Acute systemic intoxication, commonly of bacterial origin, is the most frequent cause of convulsions after the second or third year. Many of the acute infectious diseases, such as pneumonia, scarlet fever, measles, poliomyelitis, cerebrospinal meningitis, smallpox, malaria and whooping-cough, may produce convulsions. Autointoxications of the recurrent vomiting type may rarely be ushered in by convulsions. Uremia is an important cause of convulsions, especially after the third or fourth year of life, and it should always be suspected if the child is suffering from or has recently had one of the acute infectious diseases, such as influenza, scarlet fever or diphtheria. In every convulsive disorder the urine should be examined. Insolation is a not uncommon cause of convulsions in infants. The heat stroke probably acts by still further weakening the feeble inhibition of the infant. During the first days of life the most common causes of convulsions are cerebral hemorrhage, asphyxia, and birth injuries to the skull and brain.

Reflex Factors.—While the importance of reflex irritation has been greatly exaggerated as an exciting cause of eclampsia, it should be noted that these factors are quite capable of producing a convulsive seizure in highly neurotic and rachitic infants having the spasmophilic diathesis. In many of these cases the predisposing causes are so potent that the exciting causes may be almost or quite overlooked. Undigested food, worms and other irritants in the intestinal canal, and perhaps even the cutting of a tooth, may, in highly predisposed infants, be the exciting cause of convulsions. In such cases, however, the exciting causes are so slight and the predisposing causes so powerful that the latter must be considered as the important factors in producing the eclampsia.

Epilepsy.—A symptom group characterized by recurring convulsions should be suspected when convulsions are repeated from time to time without apparent cause. Convulsions may be produced by organic lesions pressing on or irritating the cerebral motor centers. Among such causes may be mentioned meningeal hemorrhage, meningitis, tumor, abscess, hydrocephalus, embolism, thrombosis, and injury to the skull or brain. The rôle, however, which these organic lesions play in producing convulsions is slight as compared with the non-organic factors previously noted.

**Symptomatology.**—Eclampsia is a syndrome and not a disease. This symptom group, however, always indicates the existence of some serious acute or chronic disease, the nature of which must be determined by other symptoms and by the general history of the case.

Certain premonitory symptoms, such as sudden twitchings of the muscles of the arms, legs, or face, associated with high fever, may indicate that eclampsia is threatened. In many instances the physician will be called, not because the patient has fever or gastrointestinal disturbance, but because the mother has become alarmed at the occasional sudden jerkings or spasmodic contractions of the legs, arms, or face of her sleeping child. It may continue to toss restlessly in its sleep for a time, and then, without awakening, pass into a general convulsion.

Perhaps in the majority of instances the convulsive storm occurs without warning. A sudden pallor of the face is followed by a convulsive stiffening of the muscles, the eyes roll up and become fixed, spasmodic contractions of muscles occur; these clonic contractions may almost immediately become tonic, producing rigidity of the entire body; the face is distorted, the head is drawn to one side, the hands are clinched upon the thumbs. Very shortly clonic convulsive jerkings of the head and extremities supervene, and these severe spasmodic movements continue for three or four minutes, leaving the child relaxed, exhausted, and in a condition of more or less profound sleep, from which it may awake bright and conscious, or, without awakening, may pass into a second convulsion. The sleep which follows may be of short duration, or it may be a true stupor or coma which continues in the interval between the convulsions. The number of convulsions in an individual case will depend upon the character and severity of the disease of which they are a symptom. There may be but one convulsion, or convulsions may be repeated at short intervals over a number of days. In the ordinary eclampsia of infancy the patient awakens from the sleep which follows the convulsion, appears bright, is conscious and gives little evidence of the severe nervous storm through which it has passed. In severe convulsive seizures incontinence of urine and feces may occur, and there may be more or less spasm of the respiratory muscles, producing a shallow, irregular breathing, or a spasmodic choking sound; cyanosis may occur and life may be threatened by asphyxia. While unconsciousness and spasmodic muscular contractions are considered a necessary part of the eclamptic syndrome, these convulsive seizures may vary greatly in severity, from a momentary unconsciousness with slight twitching of some portion of the body to a general convulsion so severe as to take the life of the child. Convulsions may involve a portion of the body, or they may be general, or again they may begin in a certain group of muscles and spread until nearly the entire muscular system is involved.

One attack of eclampsia does not necessarily predispose to another, unless some organic injury to the nervous system results from the convulsive seizure. The same predisposing causes, however, which made possible the first convulsion may account for subsequent attacks from slight exciting causes.

**Prognosis.**—Age is a very important factor in prognosis. In the new born the prognosis is bad, because convulsions at this time of life are commonly produced by asphyxia, serious brain lesions, or congenital defects,

and are, therefore, frequently followed by spastic palsies, epilepsy, and mental defects. Convulsions occurring between the second week and fourth month of life, while they are not so ominous in their import as those occurring during the first week, yet they are, as a rule, more serious than those that occur between the fourth month and the end of the second year. At this latter period convulsions may occur from comparatively trivial and easily removable causes, and the prognosis is on the whole good. In older children, especially after the third or fourth year of life, the prognosis again becomes more grave, since convulsions at this time are commonly due to uremia or to one of the acute infectious diseases. Apart from the age of the child, the prognosis may be indicated by the severity of the initial convulsion; by the depth of the supervening coma; by the continuance of localized contractures in the interval between the convulsions; by the frequent occurrence of convulsions without apparent exciting cause, by the evidence of some injury to the head, and by the presence of cyanosis, asphyxia, and spasmodic breathing. It is well, however, for the physician to remember that even the above prognostic indications do not furnish safe grounds for a positive prognosis. One should, therefore, in all cases, give a guarded prognosis and await further developments to determine the causes which have produced the convulsion, since the character of the disease which produces the eclampsia is the most important factor in prognosis. Pertussis and advanced rickets are among the etiological factors which add gravity to the prognosis.

**Treatment.**—In the majority of instances convulsions are self-limited and last such a short time that when the physician reaches the patient the convulsive movements have ceased. The treatment in these milder cases from the beginning is directed to the prevention of subsequent attacks.

In a small percentage of the cases, however, the convulsion itself is a source of danger not only to life but also to the subsequent well-being of the child, and the longer the convulsion lasts the greater are these dangers. In these cases the prime object in the treatment is to terminate the convulsion as soon as possible, regardless of its cause. This may be done by inhalations of chloroform; the convulsive movements quickly cease when a few drops of chloroform are placed upon a handkerchief and held to the child's nose. The administration of chloroform may be repeated from time to time for the purpose of cutting short the return of convulsive movements, and this treatment may be safely continued until the convulsive movements have been brought under control by other remedies. As soon as the convulsions have subsided under the inhalation of chloroform the child is placed in a bath, the temperature of which will depend upon a number of conditions. If the fever be high, one should begin with a lukewarm bath which is gradually cooled to 80°F. This not only reduces the body temperature, but exerts a soothing and tonic effect upon the nervous system. If, however, the patient be very young or very frail, the bath is not to be cooled below 90° or 95°F. Immediately following the bath an ice-cap should be applied to the head; it helps to keep down the tempera-

ture and acts as a sedative to the nervous system. The bath and ice-cap are to be used in the subsequent treatment of the case if high fever, convulsions, and other nervous symptoms are present.

A cathartic should be given as soon as possible regardless of the cause of the convulsion. The selection of the cathartic will depend upon the condition of the child's stomach. Castor oil is to be preferred if the stomach will retain it; a solution of Rochelle or Epsom salts may be substituted if the stomach be very irritable. If these be not retained, a quarter of a grain of calomel may be given every half hour until two grains are given. The importance of cathartic medication in the treatment of infantile convulsions does not depend wholly upon the fact that gastrointestinal toxemia is commonly present, since even in those cases which have their origin entirely apart from the intestinal canal free catharsis quiets the nervous system and prepares the patient for any special dietetic treatment that may be necessary in the subsequent management of the case. A high rectal enema of a pint or more of physiological salt solution should be given immediately after removing the child from the bath. The object of this treatment is to unload and irrigate the large intestine, so as to remove any possible sources of irritation and prepare it to receive and retain sedative medicines which it may not be possible to give by the mouth.

Chloral hydrate is the best and safest of all remedies to control convulsions; it should be given dissolved in starch water by high rectal enema half an hour after the colon has been unloaded by rectal irrigation. The dose per rectum for a child of six months is 5 grains and for a child of two years 10 grains; if given by the mouth these doses are to be cut in half. It is wise not to risk upsetting the stomach either with food, stimulants or other medicines until the initial cathartic has been retained a sufficient length of time to insure its action. If the chloral is retained by rectum for three-quarters of an hour and the convulsive movements are under control, the physician may safely leave the case for the time being in the hands of a competent nurse with directions that the injection of chloral be repeated in one or two hours, and thereafter it should be given by the mouth in such doses and at such intervals as may be indicated by the age and condition of the child. After twenty-four hours the dose of chloral may be diminished in size and bromid of potash in 4- or 5-grain doses combined with it; the chloral and bromid treatment should be continued until all danger of convulsions has passed.

Morphin is the most certain of all remedies for the control of convulsions. A remedy like this, however, which acts so powerfully, should be used cautiously and in the proper dosage. If the chloral is not retained by the rectum, or if the convulsion be so severe that chloral fails to control it, morphin should be given hypodermically, in doses varying from 1/50 of a grain for a child six months of age to 1/20 of a grain for a child two years of age. As a rule only one dose of morphin is necessary, and thereafter the convulsive movements may be controlled by other remedies. If, in very severe eclampsia, which requires repeated doses of mor-

phin for the control of the convulsive symptoms, a prolonged period of coma or unconsciousness should follow, it is advisable to resort to venesection followed by the injection into the vein or subcutaneous tissues of 6 or 8 ounces of sterile normal salt solution, or Fischer's solution may be given intravenously as noted under Acute Nephritis. This treatment, especially in uremic poisoning, is frequently followed by a return of the child to consciousness.

Absolute quiet for the nervous system and physiological rest for the gastrointestinal canal are necessary during the first few hours of treatment. Food and stimulants by the mouth should be avoided until the intestinal canal has been unloaded and the convulsive movements are under control. If, during this time, the child's condition demands stimulation, subcutaneous injections of normal salt solution (6 to 8 ounces) may be given at intervals of twelve hours.

Following catharsis and the control of the convulsions, water, barley water and weak beef broth may be given by the mouth, provided the child craves food or drink.

When the cause of the eclampsia has been ascertained, which may be from twelve to thirty-six hours after the onset of the first convulsion, the case is to be treated with reference to the control of the disease which caused the convulsions. If the trouble be of intestinal origin, as it commonly is in children under two years of age, then the feeding should be that of Acute Gastroenteric Infection, which is elsewhere outlined. If the trouble be due to one of the acute infections, such as pneumonia or scarlet fever, the treatment for the underlying cause must follow. If nutritional disorders, such as acute rickets, be present, and the exciting cause of the convulsion is slight, the subsequent treatment must be directed toward the removal of the profound malnutrition, which is the important factor in producing the convulsion. If uremia or other forms of autointoxication be present, the proper treatment for these conditions must be promptly instituted. If the eclampsia be due to organic disease of the nervous system, the subsequent history of the case must determine the character of the treatment.

## LARYNGISMUS STRIDULUS

(*Cerebral Croup, Child Crowing, Inward Spasms, Laryngospasm*)

Laryngismus stridulus is a reflex neurosis due to an underlying profound malnutrition. It is most commonly observed in foundling hospitals and similar institutions for the care of infants. It is caused by a spasm of respiratory muscles and especially of the adductor muscles of the larynx, which results in a sudden closure of the glottis and a temporary shutting off of air from the lungs.

**Etiology.**—Acute rickets is recognized as the most important etiological factor. Jacobi called attention to the relationship of craniotabes to this condition. Hereditary syphilis may also be an important factor. It

occurs most commonly between the sixth and the eighteenth month, and is much more frequently observed during the months of January, February and March than other portions of the year.

Stomach indigestion, enlarged cervical or bronchial lymph nodes, the dropping of mucus or other foreign substances into the larynx, fright, anger, acute adenoid and tonsillar disease, and perhaps even the cutting of a tooth, may be named among the reflex causes which are capable of touching off a laryngeal spasm in infants suffering from an advanced form of acute rickets, hereditary syphilis, or some other disease which produces a profound malnutrition and excessive irritability of the nervous system.

**Symptomatology.**—A neurotic child suffering from one of the malnutritions previously named may, with little or no warning, be seized in the early hours of the night with a spasm of the glottis, which may completely shut off inspiration. As the glottis is closing, the child sometimes in its struggles gives vent to a strident noise produced by the rushing in of air before the stricture of the glottis is complete. With the shutting off of air the child struggles for breath, its face becomes cyanotic, its head is thrown back, convulsive movements of the diaphragm occur, its body stiffens, and its life seems in imminent danger, when suddenly a loud crowing inspiration announces the fact that the spasm has relaxed and all immediate danger is over. It is the strident crowing sound that marks the close of the paroxysm which characterizes the symptom group and gives it its name. Following this strident inspiration the child breathes rapidly, is greatly excited, cries and frets, and finally falls asleep, possibly to be awakened some hours later with a second attack. General convulsions follow the laryngeal spasm in one-third of the cases. Convulsive movements of the diaphragm and other respiratory muscles are, as a rule, a part of the attack. Carpopedal spasm, which is one of the classical symptoms of tetany, is present in one-half of the cases.

Second and third attacks almost always occur within a few hours after the first attack, and, in severe cases, the child may have a dozen or more paroxysms in twenty-four hours. An attack of laryngismus stridulus may occur at any time during the day or night, but the first attack of the series most commonly occurs during the most profound sleep in the early hours of the night.

*Holding-the-breath-spells* which occur in older children are closely allied to, but not identical with, laryngismus stridulus. In this condition the spasm of the larynx is usually brought on by a fit of anger. Spasms of the larynx occur in acute laryngitis, whooping-cough and other diseases, but the clinical pictures produced are quite different from that of laryngismus stridulus.

**Prognosis.**—The prognosis is good, so far as the paroxysm is concerned. If the underlying malnutrition can be successfully treated, then the prognosis, so far as ultimate recovery is concerned, is also good. Some of the more severe cases die from asphyxia or general convulsions.

**Treatment.**—Treatment of the Attack.—The child should be taken

up, cold water dashed into its face, and a cold wet towel applied to its chest. If this does not relieve the paroxysm, chloroform may be given by inhalation. In more desperate cases intubation has apparently saved life.

Prevention of the Attack.—Following the initial attack the child for the first twenty-four hours should be kept somewhat under the influence of chloral, 1 or 2 grains every two or three hours, according to its age. After the first twenty-four hours, bromid of soda or potash may be substituted for the chloral, 4 or 5 grains every four hours for a period of four or five days; in severe cases the bromid treatment may be continued for weeks at a time.

Treatment of the Underlying Causative Condition.—This is all-important and should be followed up until complete recovery takes place; to accomplish this may require years. The special treatment indicated will depend entirely upon the character of the underlying malnutrition, whether this be rickets, syphilis, or tuberculosis; the treatment of these conditions is given in other chapters. The diet must be carefully selected to suit the age, digestive capacity and individual requirements of the patient. The child should live in the open air and have as much sunlight as possible; cod-liver oil and iron are important. When it has sufficiently recovered from its malnutrition, any disease of the nose or throat that may exist should be removed by appropriate medical or surgical treatment.

## TETANY IN INFANCY AND CHILDHOOD

Tetany is a neurosis characterized by tonic contractures of muscles. These contractures may be intermittent, but, as a rule, they are persistent and subject to exacerbations at irregular intervals. The favorite site for these contractures is the extremities. The muscles of the trunk, neck and face may also be affected.

**Etiology.**—Tetany occurs with far greater frequency in Europe than it does in America. In infancy males are slightly more commonly affected than females, the proportion being 5 to 4. Most of the cases occur during the winter and spring; this is perhaps due to the presence of rickets and other malnutritions at this period of the year. About 50 per cent. of reported cases are under two years of age. From this time on it occurs with decreasing frequency throughout childhood, but is seen again with greater frequency about the period of puberty; it may occur at any age.

It is believed that some defect in parathyroid metabolism underlies many conditions characterized by muscle spasm. Attention has been called to a possible similar etiologic relationship in tetany, myotonia congenita, paramyotonia multiplex, and myokymia.

Chronic gastrointestinal toxemia is perhaps the most important etiological factor; it is present in nearly every case occurring during the first two years of life. Rachitis is present in the majority of cases. The rickets associated with tetany, however, is not commonly of a very severe type,

and in this particular it differs from the rickets associated with laryngismus stridulus. Cases have been observed to follow measles, typhoid fever, rheumatism, and pertussis.

Reflex factors, such as undigested food, worms and foreign bodies in the intestinal canal, or adherent prepuce and adenoid growths, may be sufficient to excite a paroxysm of tetany in spasmophilic infants. The predisposing factors of tetany produce an irritability of the motor peripheral nerves; this hyperexcitability of motor nerves constitutes in these infants the spasmophilic diathesis which makes it possible for slight exciting causes to produce exaggerated tonic contractions of the muscles which they supply.

The pathological changes in the nervous system which are associated with the syndrome of tetany as it occurs in the young infant are not definitely known. G. W. MacCallum and others have shown that tetany may follow extirpation of the parathyroid glands and that the symptoms in these cases may be controlled by the administration of calcium salts. It has been inferred from these facts that some defect in calcium metabolism may be etiologically related to tetany. In fatal cases hemorrhages into the parathyroids, hydrocephalus, hyperemia and edema of the brain and inflammation of the meninges and other lesions of the nervous system have been found. These changes are, however, inconstant and it is not believed, with the possible exception of the parathyroid findings, that they have any bearing on ordinary infantile tetany. Certain it is that, whatever may be the character of the changes underlying infantile tetany, they are, as a rule, temporary, since the great majority of cases terminate in complete recovery.

The predisposing causes of infantile tetany, whatever they may be in an individual case, always cause, as Escherich and von Pirquet have demonstrated, a hypertonicity of the peripheral nerves, producing in them an abnormal excitability to the galvanic current which causes the muscle group supplied by the nerve to respond with both cathodal and anodal closing contractions to less than 5 milliampères of current. The median nerve is usually selected for this test. This condition of peripheral nerve excitability to low galvanic currents is present not only in tetany, but in laryngismus stridulus, idiopathic convulsions in infancy and other conditions characterized by muscle spasm. It is spoken of as the spasmophilic diathesis.

In children suffering from the spasmophilic diathesis, attacks of tetany may be produced by such reflex factors as undigested food, worms, foreign bodies in the intestinal canal, adherent prepuce and adenoid growths or localized muscle spasm may be produced in them, as Erb, Chvostek and Trousseau long ago pointed out, by various forms of peripheral nerve irritation.

**Symptomatology.**—The most notable symptoms of tetany are tonic muscular contractures, which occur in almost any part of the body; the most common locations for these contractures are in the forearms, hands and feet, producing the carpopedal spasms. The positions assumed by the hands and feet are characteristic; the fingers are flexed at the metacarpo-

phalangeal joints, the phalanges are extended and the thumb is drawn across the palm of the hand. The wrist is sharply flexed on the arm, and the whole hand is drawn toward the ulnar side. In the more severe cases the forearms are flexed on the arms and pressed against the sides of the thorax. In moving the elbow the resistance is not so great, or so painful, as in moving the wrist. In milder cases the shoulder and elbow joints are freely movable, while the contractures of the wrist and hand are very strong. The pedal spasm usually accompanies the carpospasm and the contractures are usually symmetrical; the feet are extended, and the first phalanges of the toes are flexed, the others extended. The foot is curved inward and the tendo Achilles is very tense. The knee and hip joints are usually freely movable; in some cases the thighs are adducted. While these contractures are commonly confined to the forearm, hands, and feet, it is not uncommon, in more severe cases, especially those under one year of age, to have contractures of muscles of the trunk and neck, producing opisthotonos and stiffening of the body. I have seen cases of this kind in which the infant's body remained rigid when lifted from the bed. In rare instances the muscles of the face and eyes are involved.

A paroxysm of tetany may continue for a few days, or it may last for weeks, and during this time the muscular contractures are, as a rule, continuous. There may, however, be periods in which there is a marked remission, or even a short intermission of the spasm. When the paroxysm has subsided, the child, under proper treatment, as a rule, progresses slowly to a satisfactory recovery. Relapses, however, may occur at variable intervals, weeks or months intervening.

Pain usually accompanies the spasm; in bad cases this may be severe enough to cause the child to cry out. Pain is greatly increased by any attempt to move the contractured part, or by stretching or pressing on the contractured muscle. There is no loss of consciousness in this disease, unless general convulsions supervene; this complication is much less common in tetany than it is in laryngismus stridulus. Edema of the feet, ankles and wrists may be present.

The increased nerve and muscle irritability finds expression in increased electrical excitability of both nerves and muscles with changes in their qualitative reaction to galvanism (Erb), (neurotonic reaction). It is also shown in the facial phenomena known as "Chvostek's symptom," in which spasm of the facial muscles is produced by percussing over the facial nerve, and in "Trousseau's symptom," where spasm of the feet and hands is greatly exaggerated by pressure upon the large nerve trunks leading to these extremities; also Hoffman's sign, an increased mechanical and electrical excitation of the sensory nerves. These phenomena, due to increased excitability of the peripheral nerves, may be observed not only during the acute paroxysm, but may be also elicited in many cases for some time after the muscular contractures have disappeared. So long, therefore, as Erb's, Trousseau's, Chvostek's and Hoffman's phenomena can be elicited, the patient is not to be considered as thoroughly convalescent from the attack.

The danger of second and third attacks or relapses is not removed until the underlying intestinal disease and malnutrition have been cured.

An elevation of temperature of two or three degrees is almost always present. This is perhaps due in part to the underlying toxemia. When the intestinal canal has been unloaded and careful feeding instituted, the temperature may fall to normal and remain so, even though symptoms of tetany may remain.

**Differential Diagnosis.**—Tetany is to be differentiated from tetanus by the locations of the contractures and by their intermittency, and especially by the absence of trismus. Trousseau's, Erb's, and Chvostek's symptoms are absent in tetanus. The age, previous history and general condition of the child will materially assist in the differential diagnosis.

**Treatment.**—Calomel followed by castor oil will serve the purpose of removing irritating and poisonous materials from the intestine and will prepare the patient for the very careful dietetic treatment that is to follow. The child must be fed with a view not only to correcting the existing malnutrition, but also to preventing further intestinal intoxication. This should be done along the lines detailed in the chapter on Chronic Intestinal Indigestion. For the control of the spasm, chloral and bromids may be used in moderate doses. Lukewarm baths, two or three times a day, will not only help in the relief of the spasm, but will benefit the intestinal condition. The child should be given sunlight and fresh air; these are almost as necessary in the treatment of tetany as they are in tuberculosis. The patient should be kept as quiet as possible and protected from noises and reflex causes of irritation. As the child improves, cod-liver oil and iron are of great value in overcoming the malnutrition. A careful search should also be made for every possible cause of reflex irritation. The prepuce and rectum should be examined, and, as the child convalesces, the throat and nose should be inspected. The removal of reflex factors may facilitate recovery. Calcium lactate in 3- to 5-grain doses may be tried if the tetany does not yield in a few days to the dietetic treatment above outlined.

## NYSTAGMUS AND ASSOCIATED MOVEMENTS OF THE HEAD IN INFANTS

W. B. Hadden, under the title, "Head-nodding and Head-jerking in Children, Commonly Associated with Nystagmus," described a not uncommon neurosis characterized by rotary, lateral, or vertical movements of the head, usually associated with rotary, lateral, or vertical movements of the eyes.

**Character of the Movements.**—Peterson described, under the term "gyrospasms," a rotary movement of the head from right to left and left to right. These head movements may also take the form of "head-nodding"; in these cases the head moves with a vertical nodding motion. In other cases the movements of the head are horizontal. These vibratory

movements are, as a rule, rhythmical and rapid, two or three vibrations occurring to the second. The same movements, however, do not always persist; any one may be replaced by or alternate with either of the others, or the three movements of the head, vertical, horizontal, and rotary, may all occur at different times in the same patient. Hadden says that pure nodding is rare, but this movement is commonly combined with or alternates with the lateral or rotary movements. In some cases these movements may cease when the child's attention is firmly fixed on some object, but, as a rule, they are increased when the child is under observation. During sleep the movements cease, and they are not so well marked and commonly disappear when the child is lying down and quiet in a darkened room, and they may sometimes cease when the eyes are covered.

Nystagmus is commonly associated with these head movements; the eye movements may be rotary, vertical, or lateral. The movements of the eyes, however, are more rapid than the movements of the head, the vibrations in some instances being as rapid as six to the second. These involuntary vibrations of the eye are, as a rule, rhythmical. The horizontal movement is the most common, but it may alternate with, or be replaced by, vertical or rotary movements, and, rarely, according to Mills, "the vertical and horizontal oscillations may alternate regularly or irregularly, or a vertical movement may be present in one eye and a horizontal in another. The commonest form of nystagmus is that in which the movement is bilateral, horizontal, and consentaneous."

The movements of the head and eyes do not always correspond. Any form of eye movement may be combined with any form of head movement; for example, head-nodding may be combined with lateral nystagmus, or we may have nystagmus of one eye associated with any form of head movement. In short, any number of combinations of the various head movements and eye movements are possible, but it should be remembered that in perhaps a majority of cases the head and eyes move in the same direction. The various head movements above described, while commonly associated with nystagmus, may occur without the nystagmus, and, on the other hand, the nystagmus may occur without the head movements.

**Etiology.**—This syndrome usually occurs during the first year of life, commonly between the second and twelfth months. During the second year of life it is not infrequent, but after that it is very uncommon, except as it is associated with ocular defects, organic disease of the nervous system, insanity, or congenital idiocy. In this chapter, however, we are interested only in this syndrome as a manifestation of a not uncommon neurosis, which occurs almost exclusively between the beginning of the third and the end of the twentieth month of life.

Heredity is an important predisposing factor. In many of the cases there is a bad neurotic family history; epilepsy, chorea, hysteria, and other neuroses, which are characterized by feeble inhibition, have been noted. Rachitis and gastrointestinal disease, with improper food, impure air, and bad hygienic surroundings, are very important predisposing causes.

**Prognosis.**—The prognosis is commonly good. This syndrome, however, in one or more of its manifestations may continue for months, but under proper care recovery finally occurs. The head movements, as a rule, disappear before the nystagmus.

In making the prognosis in an individual case it is important that the neuroses above described be carefully differentiated from the same head and eye movements occurring in certain organic diseases of the brain, as well as these same movements occurring with the so-called imperative movements of defective children. These imperative movements in feeble-minded children very commonly take the form of a salaam, or repeated movements of the arm, trunk or leg. If such movements as these are associated with the syndrome under discussion, the prognosis is not so good.

**Treatment.**—The treatment is largely a matter of improving the child's general nutrition. Rachitis and the underlying gastrointestinal disease, if present, must be treated by diet and proper medication. A carefully selected diet, suitable to the age and digestive capacity of the child, is absolutely necessary; fresh air and wholesome hygienic surroundings should be insisted upon. Cod-liver oil and some palatable and easily assimilated preparation of iron may be of value. Under this treatment the child's malnutrition gradually disappears, the nervous centers are better nourished, become less irritable, and the inhibitory centers of the cortex gradually assume more perfect control of the lower centers, and, as a result, the syndrome disappears.

Sedative medication may be indicated in beginning the treatment of some of these cases. Bromids may be given in from 3- to 5-grain doses three or four times in twenty-four hours, but they should be discontinued unless there is evidence that they are of decided value.

## EPILEPSY

**Definition.**—The syndrome which, regardless of its etiology we call epilepsy is characterized by habitually recurring attacks of loss of consciousness and loss of motor coördination, which commonly find expression in convulsive seizures either local or general.

**Etiology.**—Epilepsy is produced by a variety of pathological conditions. It may be organic, due to some defect, disease, or injury of the nervous system, or it may be idiopathic, due to reflex toxic, constitutional or hereditary factors, the influence of which is not clearly evident.

*Age* is an important etiological factor. The vast majority of these cases begin during childhood, and in a considerable percentage the early symptoms are manifest during the first year of life.

*Heredity* as a predisposing factor is present in one-sixth of all cases, and in one-third of those in which there is no evidence of organic disease of the nervous system. The family history may show epilepsy, infantile eclampsia, hysteria, insanity, migraine, or some other well-marked neurotic tendency; or there may be a history of hereditary syphilis.

*Chronic malnutritions,* especially those produced by rickets, chronic gastrointestinal disorders, and chronic heart disease, may be etiological factors. Autointoxications, especially of the migrainous type, may assist in the production of epilepsy; the association of migraine and epilepsy is noted by all authors; in these cases there is commonly a family history of gout or of the arthritic diathesis.

*Reflex irritation,* although perhaps not the most important causative factor, is closely associated with the development of many cases of epilepsy, and once the epileptic habit has been formed reflex irritation is one of the most common factors in precipitating attacks. Abnormal conditions in the intestinal canal, the eye, the nose, the throat, and the genitourinary organs are the most common causes of reflex irritation. Teething has also been mentioned as a reflex factor.

Organic epilepsy has as its essential pathological factor some *organic disease of the nervous system,* such as agenesis, porencephalia, microcephalus cysts formed by a softening of the brain substance secondary to obstruction of blood vessels, tumors of the brain and cord, traumatism producing fracture of the skull, and, most important of all, cerebral hemorrhages, especially minute punctate forceps injuries occurring as one of the accidents of birth, or traumas resulting from severe convulsions, or injury to the head in very early infancy. It had long been known that these injuries to the brain were responsible for a large number of epilepsies, but a new interest was added to this subject by the admirable clinical studies of B. Sachs, who demonstrated that many obscure epilepsies developing in late childhood were focal epilepsies having their origin in cortical hemorrhages, which occurred in infancy during or shortly after birth. In many of these cases epilepsy develops when the only remaining signs of the spastic palsy, which the original lesion produced, are increased reflexes and unilateral muscular weakness.

**Symptomatology.**—*Grand mal* is the most important clinical type of epilepsy. It is characterized by a sharp cry, loss of consciousness, a fall, and tonic convulsive movements quickly succeeded by general clonic convulsions. The convulsive movements last for a few minutes, and are followed by a more or less profound sleep, from which the patient awakens convalescent from the attack, and with little or no knowledge of what has happened.

*Petit mal,* or the minor attacks, is characterized by sudden loss of consciousness of short duration, sometimes only momentary, and by slight local convulsive movements, which may be confined to the fingers or face; these movements are often so slight as to escape attention. The patient recovers himself almost immediately and is usually conscious that an interval of unconsciousness has passed.

Between grand mal and petit mal, which represent the extreme types of epilepsy, we may have great variations in the severity of the degree of unconsciousness and of the convulsive movements, and these gradations, together with the less characteristic symptoms that mark the

individual attacks, give great variety in symptom grouping to epileptic seizures.

*Jacksonian Epilepsy.*—In these cases there is an unilateral lesion of the central nervous system which may or may not be evidenced by hemiplegia, increased reflexes, or a weakness of the muscles of one side of the body. The distinctive characteristic of this type is a monospasm which may later become hemilateral or even general. The spasm commonly begins in a local muscle group, either in the hands or face. It may be limited to this muscle group, or, as is usually the case, it may extend to one-half the body, or may result in general convulsions. There is, as a rule, no initial loss of consciousness, and consciousness may even remain unimpaired throughout the attack, except where severe general convulsions supervene.

*Nocturnal epilepsy* occurs during sleep and may not manifest itself during waking hours. Feeble inhibition is an important factor in producing this type, the convulsions occurring when voluntary inhibition is lost in sleep.

*Precursive epilepsy* occurs only in childhood. The symptoms, usually of the "petit mal" type, are associated with involuntary running movements.

*Psychic epilepsy* is very uncommon in the child. It is characterized by sudden loss of consciousness, the patient remaining motionless for a few seconds. It is not associated with convulsive movements or other symptoms of epilepsy.

*Number of Attacks.*—The habitual recurrence of attacks similar to those noted, stamps the condition as epilepsy. The number of attacks, however, may vary greatly in individual cases. Many may occur in twenty-four hours, or an interval of days, weeks or months may elapse between them.

*Aura.*—The aura includes the warning symptoms which foretell the impending attack. In focal or organic epilepsy of the Jacksonian type the aura is usually a local spasm in a special muscle group of the face, arm or leg; sensory disturbances may precede or supervene in the affected parts, and the local spasm is apt to be converted into an unilateral or general convulsion. In toxic epilepsy the aura may be vertigo, hemianopsia, or light and dark spots or flashes of light before the eyes. In idiopathic epilepsy there may be so-called sensory aura due to irritation of the cortical zones of special sense, such as a vague sensation in the stomach, a feeling of numbness or tingling in the extremities, general restlessness, irritability of temper, aphasia, or ocular or auditory phenomena.

*Loss of consciousness,* which is the most characteristic symptom of the epileptic attack, has strange variations in its manifestations. In certain cases dream-like states with partial loss of consciousness may immediately precede or follow the attack. In petit mal the loss of consciousness is often so slight that the attacks are mistaken by the laity for dizziness, fainting turns, or conscious tricks. In grand mal the unconsciousness is profound and unmistakable.

The *convulsion,* which is the next most characteristic symptom of the epileptic attack, varies greatly in severity and character. It may be so violent as to cause painful bruises and other serious injuries, or so slight that the momentary twitchings of the muscles of the face or hands may not be observed at all.

*Mental Symptoms.*—In nearly all cases of epilepsy there are more or less nervous irritability and mental impairment; in the psychic and toxic forms, however, this may be scarcely noticeable. In organic epilepsy the mental symptoms will depend upon the location and extent of the organic disease. Well-marked mental impairment is the rule, and idiocy is not uncommon. In idiopathic epilepsy there is retardation of mental development, so that the child may be considered backward or mentally deficient. In some instances there may be a peculiar cunning which enables the epileptic to commit acts of violence and so cover his tracks as to avoid suspicion. In the most aggravated cases a condition of status epilepticus or rarely hemi-epilepticus may result; it is characterized by frequent paroxysms, coma, exhaustion, elevation of temperature, rapid pulse and increased respiratory movements. These cases may terminate fatally.

**Diagnosis.**—Grand mal may be confused with hysteria, but in this latter condition the warning cry is absent; the loss of consciousness is not, as a rule, absolute; the pupils are not dilated; the eyes, instead of being turned upward and inward, stare into vacancy; there is no involuntary passage of urine and feces, and the narcotism following the attack is absent. In the diagnosis of petit mal there is more difficulty, since the mother may not accurately describe these attacks. Special importance in these cases must, therefore, be attached to the marked change in temperament and increased irritability, which have occurred since these attacks made their appearance.

Great importance attaches to the *differential diagnosis* of the various types of epilepsy. Organic or focal epilepsy is frequently mistaken for the idiopathic form, unless the physician in every case makes a careful search for organic disease of the nervous system. If Sachs' advice be followed to test in every case the comparative strength of the muscles of the right and left hand, and search for an exaggeration of deep reflexes, and, in older children, for the Babinski sign, as well as inquire carefully into the early history of the child for evidences of cerebral hemorrhage or other disease of the nervous system, many cases that have been classed as idiopathic will be found to be organic. Partial convulsions, which may or may not become general, persistent headaches, and optic neuritis may indicate organic epilepsy. Nocturnal epilepsy may exist unsuspected for a long time. In these cases the following symptoms occurring during the night are significant: biting the tongue, producing blood on the pillow, and incontinence of urine and feces, followed the next morning by lassitude, mental dullness, and headache.

**Prognosis.**—About 10 per cent. of all cases of epilepsy get well under proper treatment. In organic epilepsy the prognosis is unfavorable, unless

early operative interference is resorted to; a few of these cases, however, due to syphilis, are improved by antisyphilitic treatment. In toxic epilepsy of short duration the prognosis is much better. In idiopathic epilepsy the prognosis is more favorable when the disease has lasted but a short time, and the interval between the attacks is of long duration, and where a potent and removable reflex factor is found, such as eye-strain.

**Treatment.**—Where the aura precedes the attack a sufficient length of time to permit of treatment, inhalations of chloroform; or nitrate of amyl, may shorten or prevent the attack. During the seizure some foreign body should be placed between the teeth to prevent injury to the tongue, but no attempt should be made to forcibly restrain the violent spasmodic movements.

General Treatment.—Some epileptics may be favorably influenced by suggestion; this may be a matter of environment or of medical or surgical treatment. Temporary improvement very commonly follows almost any radical change. Surgical operation, change of locality, or any new and promising line of treatment may lengthen the interval between the attacks to months, in cases where the interval has been days or weeks. In the beginning it is important that a careful search should be made for reflex factors. Eye-strain should receive special attention, and diseases of the nose, throat and genitourinary organs should be removed by proper surgical and other measures.

In every case it is absolutely necessary to carefully study the functions of the gastrointestinal canal. Chronic indigestion and intestinal toxemia are, in a large percentage of cases, important factors in aggravating the epilepsy. Constipation must be overcome, and the diet of the patient should be selected with reference to his age, idiosyncrasies and digestive capacity; careful feeding in selected cases may accomplish more than any form of medication. In all cases, alcohol, coffee, tea, sweets, salads, pastry, and an excess of albuminous food must be avoided. In young children it is frequently necessary to diminish the quantity of fats, giving skimmed rather than whole milk.

A careful routine in the daily life of the child should be insisted upon. It should go to bed early and at a regular hour; fresh air day and night is important, and exercises suitable to the individual case should be prescribed. A quiet country life, free from noise and nervous irritation, is advisable. In mild cases mental training should be given at home, but not at school.

Chronic malnutrition and anemia, whether produced by tuberculosis, rheumatism, heart disease, malaria, disease of the digestive organs, hereditary syphilis, or attacks of influenza, or other acute infections, should receive appropriate treatment.

Medical Treatment.—The bromid of potassium is the most valuable remedy we have in epilepsy. It is not simply palliative, but when combined with the general treatment above noted it may, in selected cases, be curative. The curative effect of the bromids is probably dependent upon the

fact that the epileptic habit is interrupted, in that the irritability of the cortical sensory centers is distinctly lessened, thus giving the child an opportunity to so improve under the general treatment that after a time the bromids may be discontinued without causing a return of the epileptic attacks. In the early treatment of these cases from 30 to 60 grains of the bromid may be used in twenty-four hours, the object being to carefully graduate the dose, so that the paroxysm may be controlled without producing acne, gastrointestinal disturbances, and the general depression which may follow excessive doses. When the minimum dose of bromid has been found which will control the epileptic seizures, this dose should be continued for a year or more, and then gradually diminished during the second and third year. In nocturnal epilepsy a large dose of bromid should be given at bedtime.

The following drugs are also recommended in epilepsy: opium, tincture of belladonna, chloral, arsenic, and digitalis; the latter is indicated only when there is a complicating cardiac disease. Cannabis indica is of decided value in epilepsy associated with migraine. A warm alkaline bath followed by an alcohol rub exercises a sedative and favorable influence in many cases.

Surgical Treatment.—The surgical treatment has been on the whole disappointing, because the favorable cases do not fall into the surgeon's hands early enough to give the best results. Sachs very clearly sums up our knowledge of this subject as follows: "In a case due to a traumatic or organic lesion an early operation may prevent the development of cerebral sclerosis. If early operation is not done the occurrence of epilepsy is a warning that secondary sclerosis has been established, and an operation may prevent it from increasing. Operation must include the removal of the diseased area; here, if all other parts are normal, a cure may result. Under favorable conditions a few cases of epilepsy may be cured by surgery, and many more improved. . . . I consider it important not to await the actual development of epilepsy; and if the brain has sustained any considerable injury, to remove the injured tissues, which, if allowed to remain, constitute a permanent menace to the future health of the patient. We shall be able to prevent the development of epilepsy very much more readily than we can cure it if once established."

## CHOREA

(*Sydenham's Chorea, St. Vitus' Dance, Chorea Minor, St. Anthony's Dance*)

This condition must be differentiated from habit spasm, organic chorea, and electrical chorea.

**Definition.**—Chorea is a syndrome occurring chiefly in children, characterized by involuntary, inconstant, incoördinate, jerky, and purposeless muscular movements involving a part or all of the voluntary muscles and occurring only when the patient is awake.

**Etiology.**—Predisposing Causes.—Chorea may occur at any age; it is most commonly seen between six and fifteen; it is rare under three and a half years of age. It occurs from two to three times more frequently in females than in males. It is uncommon in the negro. It may occur at any season of the year, but is much more prevalent during the latter part of the winter and spring. The prevalence of this disease between the months of February and June is not altogether due to climatic conditions; acute infections and the nervous strain of school life may perhaps be important factors in increasing the number of cases during this season of the year. A family history of neurotic disease, gout, rheumatism, migraine, or tuberculosis is very common. Chronic anemia and chronic malnutrition are among the most important predisposing factors; children of this type have irritable, unstable, nervous systems, which make them very susceptible to functional nervous diseases of all kinds, and especially to chorea. Chronic lymph-node tuberculosis, which is the most important anemia producer of childhood, is an important etiological factor of many cases of chorea. Chronic gastrointestinal disturbances and chronic malaria may also be etiologically related to chorea.

Exciting Causes.—Toxins acting on the central nervous system are responsible for most of these cases. These toxins may be either bacterial or autogenetic, or they may be either systemic or intestinal. In the great majority of instances it is impossible to name the character or locate the origin of these toxins, and, on the other hand, it is altogether probable that there is a group of so-called idiopathic choreas associated with profound nutritional and functional disturbances of the brain, in which there may be a question as to the presence of any toxic factor.

Rheumatism is the most important cause of chorea; it is present in from 25 to 30 per cent. of all cases. This percentage may be definitely ascribed to acute rheumatic fever, whose specific cause is unknown. If, however, the term rheumatism is used loosely to designate the various forms of arthritis which follow such acute and chronic infections as tonsillitis, septicemia, tuberculosis, scarlatina, influenza, diphtheria, typhoid fever, measles, gonorrhea, and syphilis, then a much larger percentage of the cases of chorea will be found to accompany, or follow, this syndrome. All of the above-named acute infections, especially tonsillitis, tuberculosis, and scarlatina, are not uncommonly followed by chorea.

Endocarditis is present in about 25 per cent. of the cases. Cheadle and many other writers have called special attention to the relationship which exists between tonsillitis, arthritis, endocarditis, and chorea. All heart murmurs occurring during chorea are not due to endocarditis. In many instances there may be a weakening or irritability of the cardiac muscles, producing a very distinct cardiac murmur, which disappears, leaving the heart in a normal condition, when the attack of chorea has subsided. The cardiac murmurs present in highly neurotic, anemic children are very commonly not of organic origin. The common association of endocarditis with chorea should lead one to suspect organic disease of

the heart in all cases where a murmur is present, until it can be definitely proven that the heart is not diseased.

Fright, which by nearly all writers is classed as one of the important exciting causes of chorea, is responsible for the onset of the attack in a considerable percentage of cases. The fright, however, is made potent by the presence of other very important predisposing factors, such as malnutrition and general nervous irritability. Among other exciting factors one may mention intestinal parasites, gastrointestinal diseases, eye-strain, diseases of the nose and pharynx, phimosis, masturbation, delayed menstruation, pregnancy and imitation.

**Duration.**—The average duration is about ten weeks; mild cases get well in two or three weeks, and severe ones may continue for months. Cases with severe cardiac lesions, or grave nutritional disturbances, may continue for six or more months. Those that continue for years are due to organic disease of the nervous system.

**Recurrence.**—Children who have had chorea should be kept closely under observation for a number of years to prevent a recurrence. Attacks may recur at the same time of the year until the etiological factors which produced the first attack have been removed, or until age confers immunity; recurrences are not common after fifteen. Second and third attacks occur in about one-third of all cases. Children suffering from profound nutritional disturbances or from chronic diseases of the nose and throat, or from organic disease of the heart, are more likely to have subsequent attacks.

**Prognosis.**—This is nearly always good. When death occurs it is due to cardiac disease or to the organic disease of which the chorea is a symptom.

**Symptomatology.**—Characteristic Symptoms.—Before the characteristic symptoms of chorea develop, the child, as a rule, is anemic, nervous and irritable. At school the teacher may observe his inability to sit still and a clumsiness in the handling of objects. The dropping of pencils, books and other things brings reproof under which the child's restlessness increases. Later, twitchings of the muscles of the shoulder, face, or hand suggest the nature of the illness. In the early history of mild attacks the child may be able to partly control these irregular, purposeless, jerky movements, but the muscular spasm in these cases may be aggravated by directing it to perform some rather delicate movements, such as threading a needle, or lifting a pin from a smooth surface. The early awkwardness of choreic children may sometimes be noted by their tripping, stumbling gait, or by peculiar muscular contractions which momentarily distort the face. Very soon, following these early symptoms, unmistakable and more or less general choreic movements develop, and then the diagnosis may be made at a glance.

There is probably no more clearly defined, or more characteristic, symptom group than that of well-marked chorea. The involuntary inconstant, incoördinate, jerky muscular contractions, involving the whole or part of

the body and aggravated by efforts to control them, present an unmistakable syndrome. These irregular muscular movements vary greatly in severity; mild, as a rule, in the beginning, and confined perhaps to one member of the body; in a short time they extend to the whole or half the body and increase in severity, until at the end of the second week they have reached their maximum. At this time in severe cases the muscular contractions are almost constant and the whole body may be undergoing bizarre movements, which twist or distort it to such an extent that the patient may be unable to maintain an upright position. The limbs are jerked and twisted in more or less constant movement, and every voluntary effort increases these incoördinate muscular contractions. In the less severe cases the child may be able to go about as usual, and have limited control of the spasmodic muscular movements, so that he is able to pick up a pin, button his clothes, or make letters with a pencil, but all voluntary movements of this kind are made after a few moments of deliberate preparation, and then the act is carried out with great rapidity.

Speech may not be disturbed, but, as a rule, even in mild cases there are marked deliberation, hesitancy, and some irregularity. In more severe cases the choreic movements involve the tongue and muscles of the jaw, and produce an irregularity of the respiratory rhythm. In these cases the articulation is imperfect and jerky; the patient hesitates and then speaks rapidly. The control, however, of the muscles of articulation may be lost in the middle of a word or a sentence, and in severe cases articulation may be impossible. The muscles of the larynx may be involved, producing irregularity in the tone, pitch and volume of the voice; an effort to speak may produce a whisper, a barking sound, and other unusual noises. The muscles of deglutition may be affected, producing difficulty in swallowing.

In severe cases of chorea the muscles become so exhausted by constant movement that they appear to be paralyzed. There may be also an actual loss of muscular power, and, in rare instances, this may amount to paralysis. During sleep the choreic movements subside, except in the most severe cases; this muscular rest gives the tired muscles an opportunity to recover their tone.

Choreic movements, as a rule, are general. In about one-quarter of the cases they are confined to one side of the body; these cases of hemichorea do not differ materially in other particulars from those which involve the whole body.

Choreic children are usually quick-witted, irritable, emotional, and suffer from headaches and general nervous exhaustion. As the disease progresses they may become more irritable, disobedient and selfish. In very severe cases hallucinations, delirium, and even acute mania and melancholia may develop; these latter symptoms are extremely rare.

Reflexes are so variable that they are of little diagnostic importance. They are commonly normal, sometimes quickened, or they may be diminished, and, in rare instances, absent.

Well-marked anemia is a very common symptom of chorea.

Urine.—There is nothing specific in the urine findings; albuminuria and glycosuria are occasionally noted; uric acid, as a rule, occurs in excess. Herter demonstrated the presence of hematoporphyrin in the urine, both of chorea and rheumatism.

Heart Symptoms.—In every case of chorea the heart should be watched throughout for evidence of cardiac disease. A rise of temperature without apparent cause is strongly suggestive of cardiac involvement. If a cardiac murmur develops, the case is to be treated as endocarditis. Practically all of the diastolic murmurs are organic; systolic murmurs at the base near the sternum are commonly hemic and due to anemia; apical systolic murmurs usually mean endocarditis, but they may be due to myocardial insufficiency, and entirely disappear during convalescence. Osler has called attention to the fact that many murmurs diagnosed as hemic or functional are later found to be organic. Pericarditis may also complicate chorea.

**Treatment.**—General Treatment.—It should be remembered that chorea is generally self-limited, and that mild cases can, for the most part, be satisfactorily treated with little medication.

In beginning the treatment all apparent sources of reflex irritation should be removed, and, above all, the intestinal canal should receive most careful consideration. A cathartic should be given, preferably calomel, followed by castor oil. Intestinal fermentation should be corrected by proper food and medication. The diet is important in all cases; the food should be selected to suit the individual child's digestive capacity. Milk is an ideal diet, unless intestinal disease or an idiosyncrasy forbids its use. Chicken and beef in small quantities may be allowed, and cooked fruits and easily digested vegetables may be given. Coffee, tea, strong beef soups, sweets, pastries, and all indigestible food are contraindicated.

Rest, both of body and mind, is necessary. In mild cases it may be possible to get on fairly well without putting the child to bed, provided he is kept moderately quiet and not allowed to engage in childish sports with other children. In severe cases the child should be put to bed and kept there until the paroxysm commences to subside, and, thereafter, until convalescence is established, he should spend the greater portion of the time in bed. In the most severe cases the railing about the bed should be high and well-padded to prevent convulsive movements from throwing the child out of bed, or from injuring him by knocks against hard objects. The bodily rest, which is so important in the treatment of uncomplicated chorea, is even more important when there is a concurrent endocarditis. Mental rest is quite as important as bodily rest. Nervous strain and mental work should be reduced to a minimum. The tactful mother and nurse when properly directed will be able to interest the child without tiring or irritating him.

Medical Treatment.—Arsenic is the most valuable remedy we have in the treatment of the attack. In some cases it undoubtedly exercises a

controlling influence over the symptoms and shortens the duration of the disease. In giving arsenic one should commence with small doses, 2 or 3 minims well diluted with water, three times a day. After a few days, when it has been ascertained that the arsenic will be tolerated, the dose is to be gradually increased until the patient is taking 10 or 12 minims three times a day; larger doses, as a rule, are unnecessary, and it is unwise to steadily increase the arsenic until the characteristic signs of arsenic poisoning are produced. These symptoms are headache, irritable stomach, diarrhea, and puffiness of the face, and should they make their appearance at any time during the administration of the arsenic, this drug should be at once stopped for a week or ten days, and then, if continued at all, should be given in small doses. In giving arsenic as above described the maximum dose of 10 or 12 drops should be continued for a few days only, and the dose should then be gradually diminished until the patient is taking from 3 to 5 drops three times a day. After giving the arsenic for two weeks, if decided improvement in the symptoms has not resulted, it should be discontinued.

Sodium salicylate (wintergreen), aspirin, and salol may be used with advantage in rheumatic cases. Warm baths and mild laxatives, such as sodium phosphate, are of value. In cases associated with profound anemia and malnutrition, iron, cod-liver oil, and a diet of meat, eggs and milk are indicated. Quinin is of value only in those cases where there is a malarial intoxication.

Sedative medication may be indicated in very severe cases. Chloral hydrate, veronal, and potassium bromid may be used to produce sleep, and, in rare instances, hydrobromate of hyacin, or morphin hypodermically, or chloroform by inhalation may be necessary to control severe muscular contractions.

Following an attack of chorea, when the patient is thoroughly convalescent, he should have his nose and throat carefully investigated. Tonsils and adenoids enlarged by disease are open portals through which infections capable of producing chorea may enter the body; they should therefore be removed to prevent acute infections. Rheumatic cases may require a change of climate to avoid the damp, cold months of the winters in our middle and northern States.

In the treatment of those cases in which there is an underlying profound anemia and malnutrition, the syrup of the iodid of iron, cod-liver oil, fresh air, good food, and appropriate hygienic measures should be continued until the child is restored to health.

## HABIT-SPASM AND OTHER HABIT NEUROSES

Habit-spasm, or tic, is a pure neurosis characterized by sudden and quick contractions which assume somewhat the character of convulsive movements. In the beginning these movements appear to be voluntary,

but by repetition they become habitual and involuntary. They occur most frequently in the muscles of the face, neck and shoulders.

**Etiology.**—Tic is in no way related to chorea. The clinical pictures which the two conditions present, and the etiological factors which produce them, are quite different. Heredity is the most important predisposing factor. These patients, as a rule, have unstable and easily excitable nervous systems inherited from neurotic ancestors. Tic is commonly associated with other neurotic disorders. Malnutrition and anemia are frequently present. These exciting factors may be brought about by bad hygienic surroundings, improper food, chronic intestinal indigestion, chronic intoxications, systemic and intestinal, and, in fact, by any pathological condition which undermines the child's general health. Tic rarely occurs before the third year of life. It most usually has its onset between the fifth and the fourteenth years. The development of the reproductive organs and school life are important causative factors. School life brings to bear on the irritable nervous systems of neurotic children the etiological factors which are most important in the development of habit-spasm. The mental training, confinement, restraint, enforced quiet, unhygienic surroundings, anxiety to excel, and increased eye-strain which school life entails may all be factors in aggravating the neurotic tendencies of nervous children, and more or less directly lead to habit-spasm.

Imitation and reflex irritation from eye-strain, or disease of the eyes, nose, throat, and pharynx are among the common exciting causes of tic.

**Symptomatology.**—The child is nervous, restless, quick of movement, and, as a rule, bright of mind. But the characteristic symptom is recurring spasmodic movements in one or more groups of muscles, commonly in the face, neck or shoulders. In an individual case the same movements are usually repeated. There may be rapid winking or blinking of the eyes, with the drawing of the mouth downward and to the side, distorting the face. The eyebrows may be raised or lowered, as in frowning. A sudden twisting or shaking of the head and shrugging of the shoulders are very characteristic movements. A peculiar inspiratory sniff with the lifting of the alæ of the nostrils occurs in some cases. Hyperesthesia of the skin may be associated with this condition. Habit-spasm of muscle groups in arms and legs may also occur, but is not common. The spasmodic movements in tic may recur at short intervals, especially when the patient is under observation. Attention to and discussion of these symptoms increases the frequency and violence of the contractions. They may almost or quite disappear during the vacation months if this time is spent in a quiet country place, and they may reappear when the child returns to school in the autumn. The worst cases are seen in the latter part of the winter and early spring months; in these there are usually a well-marked malnutrition and anemia, and hemic and accidental cardiac murmurs may be heard over the base of the heart.

**Prognosis.**—Habit-spasm, or simple tic, may continue for many months and even years; as a rule, however, the prognosis is good, provided the

hereditary taint is not too strong and the child can be placed under the most favorable conditions for recovery. We are not here interested in the convulsive and psychical tics which may occur in older children and in adults.

**Treatment.**—The treatment of simple tic should begin with the removal of all abnormal conditions which may possibly be a source of reflex irritation; eye-strain and nasal and pharyngeal irritation should receive special attention. The child should be taken out of school and have such mental training as is thought necessary at home. It should be protected from all forms of mental excitement, and its surroundings should be such that attention would never be called to the spasm. In young children the attendants should deny, if necessary, in the presence of the child, the very existence of the spasm. In older children rewards are sometimes efficacious. Children suffering from tic should not be permitted to play with other nervous children, since the disease by imitation may be communicated to others.

An outdoor life, peaceful, quiet surroundings, well-directed exercise, a carefully selected nutritious diet, and medication such as may be indicated to relieve the particular form of malnutrition, indigestion or intoxication, which may be the basic factor in an individual case, are in every instance part of the general treatment. It may be necessary by proper medication, diet, fresh air, and general hygienic measures to treat an underlying migrainous or uric acid diathesis, or a tuberculous anemia.

Drugs are of little value in the treatment of this condition, but the bromids may be used for a time to control symptoms.

Thumb-sucking is a habit neurosis which has its origin in the animal instinct of self-preservation which causes the infant to suck everything that comes in contact with its lips. The child by instinct conveys to its mouth everything that touches its hands, and when nothing happens to be in the hand the child places its thumb, fingers or some other portion of its body in its mouth. In this way the injurious habit of almost constant sucking is gradually developed. In the beginning the act is done in response to normal instincts, but after a time the sucking habit is gradually formed, and then the infant, during the greater portion of its waking moments, indulges this habit and seems to get comfort and satisfaction from the act. When this habit is once formed the infant does not, as in the beginning, suck promiscuously anything that happens to come in contact with its mouth, but confines the habit to some particular object, such as a thumb, finger, toe, the tongue, a rubber nipple, a piece of cloth, or some special toy.

The habit of sucking does not produce any notable constitutional disturbances and does not apparently influence the growth and development of the nervous system, and the infant is allowed to form this habit because the mother or the physician does not believe it is worth while to try to prevent the formation of a habit which gives the child a pleasurable occupation and does not seriously interfere with its development. The

sucking habit, however, does produce certain deformities of the part sucked, and may also lead to irregularities in the development of the mouth. The deformities of the mouth, thumbs and fingers may, in aggravated instances, be so pronounced that they are noticeable when the child grows up. It is for the purpose of preventing these deformities that the sucking habit should be corrected.

If the child is allowed to indulge in this habit for months or years, it may be necessary to use some mechanical device which makes it impossible for the child to continue it. When the habit has been indulged in for only a short period of time, it may be possible to overcome it by covering the thumb, fingers, or parts sucked with bandages or mittens carrying solutions of quinin or aloes. These bitter solutions, however, are of little value when the habit is well formed. The mechanical means which may be used may vary with the individual child and with the part of the body sucked. Punishment, as a rule, does not favorably influence this habit, but rather teaches the child deception. Older children may be influenced by rewards or by appealing to their sense of shame. The sucking habit is always more difficult of treatment in nervous, malnourished children, and, for this reason, malnutrition and other causes of nervousness should be carefully treated before an attempt is made to break up the habit of sucking by mechanical restraint.

## PICA

Pica, or dirt-eating, is a habit neurosis which manifests itself in a curious perversion of appetite. The infantile type of this disease, in which we are here interested, commonly begins before the second year of life and tends to spontaneous recovery before the fifth year. The animal instinct of self-preservation which causes the infant to put everything it touches into its mouth is the most important factor in starting this neurosis. It occurs most commonly in neurotic, malnourished children, and is very frequently associated with gastrointestinal disorders. In older children imitation is an important factor. Functional disturbances of the stomach, which produce a burning, gnawing sensation that is relieved by the eating of dirt, chalk, Fuller's earth, and other absorbents, may be important and troublesome factors in developing and continuing this habit neurosis. Whatever may be the predisposing or exciting causes which have been active in starting the practice of dirt-eating, the habit which is thus formed becomes the important etiological factor which impels the patient to continue to satisfy this perverted appetite.

**Symptomatology.**—The strange perversion of appetite in these children causes them to forego wholesome, appetizing food for such innutritious and indigestible things as dirt, plaster, sand, gravel, chalk, Fuller's earth, clay, ashes, cinders, coal, soapstone, slate-pencils, paper, rags, hair, and sometimes such disgusting materials as their own excrement. In some instances children will refuse to take all foods except sweets, such as candy and

sugar; this sugar-eating habit may lead to dirt-eating, and the development of troublesome and disgusting types of pica. Many patients who practice the habit of dirt-eating may take for a considerable time a sufficient quantity of nutritious food; as the habit develops, however, the tendency is to gradually increase the quantity of dirt taken and diminish the quantity of food. Such patients may become anemic, malnourished and emaciated, and may develop intestinal disorders.

**Prognosis.**—This is good. The great majority of these cases recover under proper treatment before the child is three years of age. A few of them continue into the third or fourth year, and neglected cases may develop into the more severe forms of pica seen in late childhood and adult life.

**Treatment.**—The first step in the treatment is to place these patients under such supervision that it is absolutely impossible for them to continue the habit; if the habit is forcibly broken it gradually loses its hold upon the nervous system, and this measure is, therefore, a curative one. The next important step is to prescribe a proper dietary, suitable to the age and digestive capacity of the patient. The food problem is especially important, since the dietetic treatment usually comprehends the removal of some gastrointestinal irritation. In cases where food is absolutely refused (anorexia nervosa) it may be necessary to feed by gavage. Alkalies such as bicarbonate of soda or benzoate of soda exert a favorable influence in these cases; they may perhaps act by neutralizing fermentation and other acids in the stomach, and thus correct one cause of the perverted appetite.

## HYSTERIA

**Definition.**—Hysteria is a psychoneurosis due to functional disturbances of the cortical centers. It is characterized by defective will power, emotional excitability, and the control of the body and mind by perverted notions and fixed ideas, which are not uncommonly produced by suggestion.

**Etiology.**—Hysteria is a real, not a simulated, disease. Its most important etiological factor in the child is feeble inhibition; this lack of control results in apparently insignificant causes producing exaggerated motor and psychic phenomena. Hysteria is more common in the adult than in the child, but is not infrequently seen in late infancy and early childhood, and is very common between the ages of ten and fifteen. Heredity is a very powerful predisposing factor; a strong neurotic taint is commonly present. There may be a family history of hysteria, chronic alcoholism, epilepsy, insanity, chorea, or general nervous irritability. The worst cases occur in families that are mentally degenerate. There is a marked preponderance of females, but this is not so great in children as in adults.

Malnutrition is one of the important direct causes of hysteria in chil-

dren. The term malnutrition is here used very broadly, not only to include innutrition and partial starvation of nerve cells, which result from lack of sufficient food and bad hygienic surroundings, but it also comprehends the condition of nerve cells which results when they are fed with blood containing auto, intestinal or bacterial toxins. Not only poor blood, but bad or poisoned blood, is important in the etiology of hysteria in infancy and childhood.

Environment is a very important exciting cause. Hysteria is more common in the city than in the country, not only because of impure air and bad hygiene, but also because of the noise, the rush, and the strain of life in a large city, where the child is subjected to constant excitement and increased mental activity. The strain of school life and school examinations, the lack of home discipline, which allows self-indulgence and free play to the emotions, and the close association of members of a neurotic family may all be factors in the development of this disease. Great grief, emotional excitement, great physical or mental strain, nervous shock, and sudden fright may develop hysteria in those predisposed to this disease by malnutrition or heredity. Severe reflex excitation, such as may come from eye-strain, intestinal disorders and phimosis, are important factors. Diseases of the genital organs may produce hysteria in infants and young children. Suggestion is one of the most potent factors in developing symptom groups in hysterical patients. Syndromes may be suggested by the questions of the examining physician or by the story of another patient's symptoms and sufferings.

**Symptomatology.**—Extreme selfishness and dependence are common manifestations of hysteria. The defect in will power makes the patient dependent upon those around her. She is often controlled by fixed ideas with reference to her inability to think or act for herself. One of the most peculiar and characteristic examples of the control which fixed ideas have over hysterical patients is shown in the symptom group known as *astasia-abasia.* This is one of the most common of hysterical manifestations in the child, and, whatever may have been its origin, it is continued by reason of the fact that the patient has a fixed idea in her mind that she can neither stand nor walk. She may have control of her legs when lying down, moving them at will in any direction, but the minute she is placed upon her feet her legs give way, or they stiffen and she loses her equilibrium, or she may stand upon her feet and not be able to walk, making incoördinate movements of the legs when she attempts to do so.

*Paralysis,* which is a common manifestation of hysteria in the adult, is comparatively rare and of a milder type in young children. It may be flaccid and associated with diminished reflexes, but it is commonly spastic, associated with contractures and exaggeration of deep reflexes. *Anesthesia,* which is so common in the adult, and often associated with motor paralysis, is comparatively rare in the child. The paralysis and anesthesia of the hysteria of childhood do not differ from these symptoms as they occur in the adult, except that they are less frequent and less intense.

*Painful and contractured joints* occur not infrequently, even in very young children. These hysterical manifestations may be mistaken for tuberculosis or other organic disease. Hysterical aphonia occurs in children. This symptom is very commonly associated with globus hystericus, and a persistent dry, hysterical cough. Hysterical eclampsia is rare in young children, but may occur in older children, and presents the same characteristics as it does in the adult.

*In the emotional forms* of hysteria fits of crying and laughing may follow each other without apparent cause. These patients are moody, irritable, and are easily thrown into states of great nervous excitability. In extreme cases catalepsy, lethargy, trance, ecstasy and even acute mania may occur; these latter symptoms, however, are very rare in the child.

*Anorexia nervosa* is a classical symptom group which is very common even in young children, and it may occur in infancy. In this condition the patient may go for weeks without being seen to retain any food; the sight of food may produce nausea, and all food taken may be vomited, sometimes with a conscious effort. The severity of this symptom group may vary greatly; in young infants it may take the form of lack of appetite, so that all food is refused. In these cases it may be necessary to resort to gavage to prevent loss of weight and serious malnutrition.

The *urine* during hysterical attacks is light colored, of low specific gravity, and is commonly passed in large quantities. Anuresis may also occur.

**Treatment.**—In beginning the treatment of hysteria it is important that all causes of reflex irritation to the nervous system should be removed. Eye-strain, diseases of the nose, throat, reproductive and genitourinary organs should receive appropriate treatment.

The next step comprehends the removal of the underlying causes of the chronic anemias and malnutritions so commonly found in hysterical patients. In order to do this it is not only necessary to prescribe medicines, such as iron, cod-liver oil, arsenic, quinin, or some tonic that will stimulate the appetite and improve digestion, but it is of even greater importance that the diet and hygienic surroundings should be carefully adapted to the needs of the individual case. Alcohol, tea, coffee, sweets, salads, pastries, and rich and highly seasoned dishes should be avoided, and a diet prescribed which is simple, wholesome, nutritious and suitable to the digestive capacity of the patient and the malnutritions from which she suffers. The hysterical patient should live as much as possible out of doors, away from the whirl, noise and excitement of a city, and moderate exercise and congenial surroundings should be insisted upon.

Removal from the nervous atmosphere of a neurotic household, stopping of all mental stimulation, and avoiding nervous excitement are important factors in the cure. The hysterical patient should, if possible, be placed under the care of a nurse whom she loves and in whom she has confidence. This attendant should be of good physique, of sober mind, and full of tact, and she should have sufficient intelligence to study the pe-

culiarities of her patient's mental condition so that she may tactfully avoid touching upon topics which by suggestion may influence her patient unfavorably; she should also be able to utilize the fads and idiosyncrasies of her patient in such a way as to prevent her from dwelling upon her troubles. The successful treatment will depend largely upon the ability of the physician to so control the surroundings of his patient that she will be constantly influenced by wholesome suggestions—suggestions that she is improving from time to time, and that her early recovery is assured. The influence of change is so important that it is necessary that radical changes should be made in the surroundings from time to time. In older children a modified Weir Mitchell rest-cure is often of great advantage. The confinement to bed, massage, forced feeding, isolation, and striking change of surroundings, which this treatment comprehends, act not only by suggestion, but the treatment itself exercises a curative influence. Hydrotherapy in some form is applicable to the treatment of nearly every case of hysteria; the cold tub bath, or cold douche to the spine, may be used in older children to control severe paroxysms. In the great majority of cases a warm bath followed by general massage and an alcohol rub is of great advantage. Electricity is a therapeutic agent, which acts largely by suggestion; in the treatment of aphonia and paralyses of various kinds it is especially valuable. Blistering the skin over tender areas and cauterizing the sensitive spine may be of benefit in some cases.

Sedatives, such as the bromids, valerian and asafetida, may be used for the relief of nervous symptoms, but they should not be continued for any great length of time.

## HEADACHES

**Etiology.**—Headaches are uncommon in children under five years of age, but when they do occur they are much more important in their pathological significance than when they occur later. After five years of age headaches are more frequent, and become common between the ages of eight and fourteen, but even during this period they are nothing like so common as they are between the ages of twenty and forty.

Heredity is an important predisposing factor; this is especially true of migrainous, neurasthenic, and neuralgic headaches. These cases usually have a family history of gout, migraine, neurasthenia, hysteria, or general nervous instability. Feebleness of constitution due to chronic diseases in the parent may be inherited by the child and predispose it to reflex, toxic, anemic and other varieties of headache.

Anemic Headache.—Malnourished, anemic, neurotic children may have headaches from very slight exciting causes, and the pain is commonly frontal, or vertical. Any chronic disease which produces malnutrition and anemia results in such instability and irritability of the vasomotor nerve centers that headaches may be produced by nervous excitement, nervous and physical fatigue, nervous shock, fear, anger, mental overwork,

the strain and confinement incident to school life, and by all kinds of toxic and reflex factors capable of acting upon the nervous system.

Reflex headaches are very common in childhood, especially between the ages of six and fourteen. They may be due to eye-strain, adenoid growths, undigested food, and other reflex causes. Ocular defects are very common in children of school age, and are responsible for a large percentage of the chronic headaches from which school children suffer. These headaches come on after using the eyes and grow worse toward the close of the school day; they are usually frontal or occipital and associated with general nervous irritability.

Toxic headaches of gastrointestinal origin are common in childhood and may be associated with nausea, vomiting, flatulency, diarrhea, or constipation. The diagnosis of headaches of this character may be confirmed by the relief which follows cathartic medication and careful dietetic treatment. In children over six years of age they may be more or less chronic, persisting for weeks, and the intestinal toxemia during this time may be overlooked. In this type the presence of indican and indolacetic acid in excess in the urine may call attention to their intestinal origin.

Toxic headaches are also produced by systemic bacterial poisons acting on the nerve centers. This type occurs in all the acute infectious diseases and is especially severe in influenza. Periodic headaches, neuralgic in character, are very commonly due to influenza or malaria; the supra- and infraorbital nerves are frequently involved and remain sensitive to touch in the interval between the attacks of pain. In older children infections involving the antrum of Highmore, frontal sinus and other bony cavities of the face may produce a persistent, periodic pain in the facial nerves, which may be mistaken for malarial or influenzal neuralgia.

Autotoxins such as occur in uremia may produce severe toxic headaches. Uremic headaches, however, are much less severe in the child than they are in the adult. They are commonly occipital and are associated with vertigo, nausea, vomiting and the urine findings of acute Bright's disease. Migraine, the most common form of headache produced by autotoxins, is elsewhere described.

During infancy and early childhood disease of the internal ear is the most common form of pain in the head.

Organic headache may be due to meningeal inflammation, tumors of the brain, cerebral abscess, and injuries to the brain and skull. Headaches of this character are severe, persistent, localized, and accompanied by other signs of organic disease of the brain or its membranes.

**Treatment.**—The successful treatment of headaches in childhood must be based upon a careful differential diagnosis of the various etiological factors and their relative importance. A search should first be made for reflex factors, giving special attention to eye-strain. If such causes be found, they should be removed by appropriate treatment. Attention should next be directed to the gastrointestinal canal. It is good practice to begin the treatment of all kinds of headaches with some form of cathartic

medication, such as calomel, followed by castor oil. This will clear the intestinal canal and assist materially in determining the importance of the rôle which gastrointestinal factors play in producing the headache. If the result of this treatment, the character of the headache, and the nature of the gastrointestinal discharges justify the diagnosis of toxic headache of intestinal origin, then the further treatment will consist in such diet and medication as will remove the causative condition. If, however, the headache is produced by some acute bacterial infection, such as influenza, it may be relieved by cathartic medication, the application of cold to the head, and the specific treatment of the infection of which it is a symptom. In these cases one is justified in using sedative medication to relieve the pain. For this purpose the bromids of sodium and potash, citrate of caffein, phenacetin and antipyrin may be given in doses suited to the age of the child. The coal-tar products, however, are to be recommended only in the treatment of acute conditions, and should be continued only for a short time; in chronic or recurrent headaches their continued use may do harm.

When the exciting cause of the headache is some emotional or nervous excitement brought on by fear, anger, or nervous shock, or when the headache is associated with extreme nervous irritability, or other hysterical or neurasthenic symptoms, an ice-bag to the head and good-sized doses of bromid act kindly in its relief.

Periodic headaches may be relieved by good-sized doses of quinin given in the interval between the attacks of pain, or the following combination of quinin, arsenic and iron may be used. It is of special value not only in periodic, but also anemic, headaches:

℞ Quininæ sulph. ..... 3 ss
Ferri reducti ..... 3 ss
Acidi arseniosi ..... grs. ss
M. Make capsules; number 20.
S. One after eating for a child eight to ten years of age.

It should be remembered that even after the headache has been removed by any of the above-named measures there may yet remain to be treated the constitutional causes of the general nervous irritability which was the important predisposing cause of the headache. This treatment comprehends not only fresh air, proper exercise, suitable food and well-directed medication, but also the intelligent direction of the whole life of the child, so that he may be properly nourished, his constitutional and local diseases eradicated, and his nervous system so protected that it may recover its normal tone and powers of resistance.

## ASTHMA

Asthma is a neurosis characterized by recurrent attacks of spasmodic dyspnea, or sibilant bronchitis, usually associated with, or followed by, a discharge of mucus from the bronchial tubes.

**Etiology.**—The underlying causes of asthma are not definitely known, but it is a well-established fact that in this condition there is a definite specific underlying predisposition which makes it possible for a great variety of exciting causes to produce an asthmatic attack. The specificity of the underlying predisposing cause is demonstrated by the fact that the same exciting cause is nearly always present in an individual case, and that the many exciting factors which precipitate attacks of asthma in certain individuals may in others be altogether impotent in producing an attack.

The bronchostenosis which occurs in all cases of asthma is believed to be due, in some cases, to a vasomotor disturbance producing a swelling of the mucous membranes or of the submucous tissues of the bronchioli, but in the great majority of cases it is due to a tonic contraction of the muscle fibers of the smallest bronchial tubes.

Asthma is much more common in adult life, but it may occur in infancy and early childhood; sibilant bronchitis is relatively more common in childhood. Typical attacks of spasmodic asthma become more common after the sixth year of life, and increase in frequency between this period and adolescence. Heredity is an important factor; there is nearly always a family history of neurotic disease or gout. Autotoxins of the gouty or lithemic diathesis may precipitate attacks of asthma. Comby classes among the respiratory manifestations of lithemia in childhood spasmodic coryza, sibilant bronchitis, and asthmatic attacks (see Recurrent Coryza). Intestinal toxemia is an important exciting cause in children. Asthmatic attacks may be precipitated by constipation, overloading the stomach, intestinal indigestion, and gastrointestinal disturbances of various kinds, or they may precede or follow urticaria of the skin. Diseases of the respiratory tract, such as catarrh of the nasopharynx, hypertrophied turbinated bones, enlarged tonsils, adenoids, bronchitis, whooping-cough, influenza, and measles, may be exciting causes.

In especially susceptible individuals the pollen of certain grasses and of rag-weed, emanations from animals, such as the dog, horse, cat, or guinea-pig, the aroma of certain medicines, and the odor of certain flowers may be specific exciting causes. Among other exciting factors may be mentioned dust, irritating vapors, fright, and atmospheric and climatic conditions.

**Symptomatology.**—Asthmatic attacks resembling the adult type, while comparatively rare in the infant, are not infrequent in older children. They may recur at irregular intervals, weeks or months intervening. The severe dyspnea which characterizes these attacks may recur nightly for a time, or in other instances may continue with marked severity for twenty-four or thirty-six hours, and then gradually subside into convalescence. Typical attacks of asthma, as a rule, begin suddenly in the night with a wheezing respiration, which soon becomes a marked dyspnea. The child sits up in bed, fixing his shoulders or arms so as to bring all the accessory muscles of inspiration into play in the attempt to force air through

the contracted bronchi into the already distended air vesicles. Emphysema develops and gives a barrel-shaped appearance to the chest in the later stages of the attack. Expiration is prolonged and accompanied by sonorous wheezing râles; the vesicular murmur may be inaudible. After a number of hours the dyspnea gradually subsides, and is, as a rule, followed by a cough with wheezing large moist râles, and more or less mucous expectoration. These symptoms may continue for a few hours or for days and subside into convalescence.

In infants and young children afebrile sibilant bronchitis with slight dyspnea is much more common than the asthmatic paroxysm above described. The dyspnea in this condition may not be very great, but the number of respirations is markedly increased, and sibilant, wheezy bronchial sounds occur, which may persist for five or six weeks. During this time these patients have no pain, suffer comparatively little discomfort, go about the house and amuse themselves without complaining of feeling ill.

La Fétra calls special attention to the eosinophilia which occurs in asthma. He says: "The leukocytes are usually, but not always, increased as in bronchitis, but a differential count of the white cells shows, what does not occur in bronchitis, a constant and usually marked increase in the number of polymorphonuclear eosinophiles (16 to 18 per cent.)."

**Prognosis.**—Patients rarely die from asthma, and the prognosis, so far as ultimate recovery is concerned, is also fairly good, provided they are so situated that they can take advantage of the means offered for its cure. Chronic cases, which have gone on to the development of chronic emphysema, do not yield readily to any form of treatment.

**Treatment.**—Treatment of the Attack.—Fresh air and the inhalation of the fumes of stramonium leaves and niter paper may relieve the paroxysm; chloroform by inhalation will temporarily arrest the attack. In older children 1/10 of a grain of morphin given hypodermically may be used to terminate the paroxysm. Atropin, 1/1,000 of a grain, with nitroglycerin, 1/300 of a grain, given hypodermically, has a favorable influence in controlling the paroxysm; if necessary this dose may be repeated in two or three hours. An emetic will sometimes cut short a paroxysm of asthma, even when the gastric contents have little to do with exciting the attack; syrup of ipecac may be used for this purpose. Tincture of belladonna, or atropin, combined with bromid of potash, chloral, or antipyrin, in doses suited to the age of the child, is a valuable remedy for modifying, shortening or preventing an attack.

Asthmatic attacks due to swelling of the bronchial mucous membrane may be cut short by local applications to the respiratory passages of a solution of cocain and adrenalin chlorid. The 1 to 1,000 solution of adrenalin chlorid may also be used hypodermically in 1 to 3-minim doses; this remedy at times acts specifically in controlling attacks of asthma.

The Interval Treatment.—Adenoids, large tonsils, nasal hypertrophies, and all diseases of the nose and throat should receive appropriate

treatment. Bronchitis, whooping-cough, measles, influenza, and all diseases which produce catarrh of the bronchial mucous membranes should be studiously avoided, or, if present, should be carefully treated until all bronchial irritation has disappeared.

If the patient has a well-marked lithemic history, the interval treatment should be similar to that recommended in the chapter on Migraine. If no such history exists, or if the patient fail to respond to this treatment, iodid of potassium or syrup of hydriodic acid should be given over a long period of time. In many cases iodin medication is very valuable in helping to bring about a cure, and should, therefore, be given a trial in every case in which some other special form of tonic medication is not especially indicated. Cod-liver oil, iron, arsenic, and tincture of nux vomica are of value in many cases, and quinin is indicated in such as have previously suffered from malaria. All medicines which diminish the appetite, or produce gastrointestinal disturbances, are to be avoided.

Asthmatic attacks associated with urticaria of the skin should be treated as outlined under Urticaria.

*Change of climate,* or change of locality, is the most important factor in the relief and cure of asthma. But in this respect it is difficult to lay down rules, since asthmatic patients, above all others, have the strongest idiosyncrasies with reference to certain localities and certain climates; a climate or locality that may benefit one may fail to give relief to another. These patients, as a rule, do well in high and dry altitudes, unless they have chronic emphysema. Experience alone will determine the best locality for the individual asthmatic patient. It is a good rule, however, to avoid the locality in which the attack developed, especially at the season of the year when attacks are liable to occur. If the attack has developed in the city, a change to the country is advisable, and vice versa. If the attacks are worse in winter, or if they are precipitated by recurring attacks of bronchitis, it is advisable to spend the cold, damp months of the year in some such climate as that of northern California or Florida.

It may be well to note that patients suffering from an asthmatic constitution are not good subjects for the serum treatment commonly used in certain acute infections, such as diphtheria and sepsis. Sudden prostration and sometimes death may result from the use of serum in these cases.

# SECTION XII

## *DISEASES OF THE EAR*

## CHAPTER LXXXIII

### OTITIS MEDIA AND MASTOIDITIS

### OTITIS MEDIA

**Etiology.**—This disease is more common in infancy and childhood than later in life. The reasons for this may be found in the fact that the Eustachian tube at this time is shorter, larger, more patulous and opens lower in the pharynx than in the older child, and that the acute infectious diseases and acute pharyngitis, of which otitis media is a complication, are more frequent during this period.

The essential cause of otitis media is an infection of the middle ear with bacteria which have found their way from the pharynx through the Eustachian tube. Staphylococci, streptococci, pneumococci, and influenza bacilli are the most common microorganisms producing this condition. Diphtheria, tubercle, typhoid and the pyocyaneus bacilli may occasionally act as exciting factors. Some of these microorganisms are usually found on the normal mucous membranes of the nose and pharynx, or in the crypts and fissures of chronically enlarged tonsils and adenoids. This is one of the important reasons why otitis media is such a common complication of acute pharyngitis, tonsillitis, influenza, epidemic grippe, measles, scarlet fever, diphtheria, pneumonia, bronchopneumonia, whooping-cough, gastro-enteritis, congenital syphilis, and typhoid fever. In fact, any disease which lights up an acute inflammation of the nasopharynx, or which greatly reduces the vitality and resisting power of the individual child against catarrhal diseases, may be complicated or followed by an acute otitis media. Some of the microorganisms, such as the influenza bacillus and the pneumococcus, which are distinctly related to definite acute infectious processes, may have a special predilection for producing otitis media. When infection is present in the pharynx this disease may rarely be produced by swimming under water, by douching, and by blowing the nose. In such cases the infection is forced from the pharynx through the Eustachian tube into the middle ear.

**Symptomatology.**—Otitis media is almost always a secondary disease.

It is frequently masked by the infection of which it is a complication, and its onset may be obscured or modified somewhat by the presence of fever, pain and other symptoms due to other causes. In the majority of instances, however, the onset is announced by a sudden and marked rise of temperature following an attack of influenza or some other acute infection. The temperature in a few hours may reach 104° or 105°F., and is usually associated with more or less earache, indicated by the crying and fretfulness of the child. In some cases, however, acute symptoms are absent, and the first manifestation of the disease is a discharge from the external ear. This form of onset may occur in very young infants and in older infants suffering from congenital syphilis, chronic glandular tuberculosis, or chronic gastrointestinal disease. In these cases there may be no fever and no pain during the course of the disease, or the fever without the pain may appear following the perforation of the eardrum and the discovery of the discharge from the ear. In other cases a high and remittent fever may be present for days without any pain or other symptoms to call attention to the ear. In view of this fact, therefore, it is a wise precaution to carefully examine the eardrum and pharynx of every child suffering from obscure fever. Otitis media and mastoiditis are among the most *common causes of unexplained remittent and intermittent fevers* in young children. In rare instances these temperatures may continue for many weeks before there is marked evidence either in the eardrum or over the mastoid process of middle ear or mastoid infection.

The course of the fever in otitis media is variable. It commonly continues until the eardrum is perforated by the pressure of the fluid from within or by operative measures from without. Following the perforation and the free discharge of pus the temperature falls, and if the drainage from the internal ear remains good the temperature remains at or near normal. In such cases, however, a secondary rise in temperature commonly means either the blocking up of the opening in the eardrum or the extension of the disease to the mastoid cells. The subsequent course of the temperature in such cases will depend, on the one hand, upon reestablishing the drainage from the middle ear, and, on the other, upon relieving the inflammation in the mastoid cells by operative or other measures.

Earache in older children is very common, and only in the minority of cases is it a symptom of otitis media, but when it continues at intervals for more than twenty-four hours and is associated with an unexplained elevation of temperature it indicates middle-ear inflammation. It is difficult in a child not old enough to locate pain to determine the existence of an earache, but it may be suggested by unexplained irritability, sleeplessness and paroxysms of crying, and sometimes the child by its position protects, or with its hand reaches for, its ear.

**Otoscopic Examination.**—The diagnosis of otitis media is made by discovering a discharge from the ear or by a careful examination of the eardrum. The difficulty, however, of determining the existence of an otitis

media from an examination of the eardrum is ofttimes so great that a specialist should be called upon to determine the significance of the findings of such an examination. The child with its arms at its side should be wrapped in a sheet, and its body and head firmly held by an assistant. The operator then draws the auricle downward and backward and inserts into the ear a speculum of proper size and shape, then with a head-mirror, or an electric head-light, he can, by illuminating the canal, bring the eardrum into view. It may be necessary before making this examination to cleanse the external auditory canal with a fine cotton-wrapped probe. In cases of otitis media the drum above Shrapnell's membrane may be found congested, reddened, and sometimes bulging slightly outward. If perforation has already occurred, the opening may commonly be seen in the posterior quadrant.

**Prognosis.**—The prognosis is, as a rule, good. This is especially true if the opening in the eardrum is made early in the disease. Where mastoiditis occurs as a complication the prognosis is much more serious. If the middle-ear inflammation continues through lack of proper drainage and other surgical treatment, hearing may be impaired, and, in some instances, entirely lost.

**Prophylaxis.**—In the treatment of all diseases in which otitis media is a common complication, earache should receive prompt and careful attention, and the eardrum from time to time be inspected. Children who have enlarged tonsils and adenoids, and who have suffered from earache or from one attack of otitis media, should have these diseased tissues removed after the acute attack has subsided.

**Treatment.**—Earache, which is commonly an early symptom, may sometimes be relieved by hot irrigations of normal saline solution. This may be accomplished by inclining the child's head over its shoulder and introducing into the ear a small straight glass medicine dropper attached to the hose of a fountain syringe. If the bag holding the hot saline solution is held one or two feet above the child's head, a steady stream of hot salt water may in this way be directed into the ear. Following this, hot flannel, hop-bags, or water bottles may be applied. If these measures fail to give relief, a few drops of warm paregoric may be dropped into the ear, and the ear plugged with warm cotton-wool. In some instances the pain may be so great that paregoric is indicated internally; it may be given in from 5- to 15-drop doses, according to age. If the earache continues, and especially if it be associated with fever, paracentesis should be performed, even though the eardrum presents little indication of the inflammation within. Following the incision a serous, seropurulent, or purulent discharge makes its appearance, sometimes at once; in other cases it may be delayed for twelve or more hours. The character of the discharge may depend upon the stage of the inflammation; in well-marked cases which have been developing for a number of days it is always purulent. During the acute stage the child should be confined to bed and the external auditory canal frequently irrigated with a warm mild saline antiseptic; follow-

ing this the auditory canal should be dried with a cotton-wrapped probe, and a small roll of antiseptic gauze inserted. A piece of gauze over the external ear and a bandage to hold this in place complete the dressing. The gauze in the auditory canal absorbs the discharge and prevents irritation and inflammation of these external parts. These details are most important in treating cases where the discharge is irritating or where it lasts over a number of weeks. If the child is otherwise in good health and the drainage from the internal ear is satisfactory, the discharge should cease within a week or ten days, and complete recovery follow. In cases, however, associated with syphilis, tuberculosis, or other forms of chronic malnutrition, the otitis media usually continues until these underlying causes have been removed by proper treatment. In such cases of prolonged middle-ear suppuration there is great danger that the hearing may be impaired or lost.

## MASTOIDITIS

**Etiology.**—Since this condition is nearly always secondary to acute or chronic otitis media, its causative factors are the same as those just outlined for that disease. The inflammation spreads from the middle ear into the mastoid cells, and causes inflammation and necrosis of these tissues. In every case of otitis media the physician should constantly be on the lookout for the development of a mastoiditis.

**Symptomatology.**—Associated with the symptoms of an acute otitis media we may have, as indications of a mastoiditis, an unexplainable rise in temperature. That is to say, careful otoscopy may show that the opening in the drum membrane is sufficiently large for drainage, and may indicate that the middle ear condition is improving, and yet, notwithstanding these facts, a septic temperature develops, which may run to 104° or 105°F. some time during the day, and fall below normal at another. In another class of cases, when a free incision of the drum membrane followed by a copious discharge of pus fails to clear up the septic temperature, mastoiditis is to be suspected. The diagnosis is confirmed by finding the tissues over the mastoid slightly reddened, swollen, and tender to the touch. Tenderness above the tip of the mastoid can usually be elicited, even if other symptoms are absent. In making pressure care should be taken to press backward, so that the soreness which comes from the middle-ear inflammation will not be mistaken for that due to mastoiditis. In many of these cases the swelling, pain and tenderness in the mastoid region are so marked that the diagnosis can scarcely be overlooked; in others, however, the onset may be so sudden and so violent that extensive necrosis of the bone has occurred before the physician has even suspected the presence of otitis media.

There is another group of cases very obscure and very insidious, characterized by septic temperature, which may run for weeks without there being any local evidence whatever of otitis media or mastoiditis.

**Treatment.**—The treatment of the otitis media, of which the mastoiditis is a complication, should be continued as above outlined. Free drainage from the middle ear must be maintained. Blood may be withdrawn from the mastoid region by leeches, and cold applications should be made with small ice-bags. By blowing out the middle ear through a catheter, introduced into the Eustachian tube, one can more effectually increase the drainage; this operation, however, can be done only in older children. If under these measures the septic temperature and the pain and tenderness do not begin to subside, the radical operation for mastoiditis becomes necessary. Chronic mastoiditis, which fails to yield to proper surgical treatment, may have as its underlying etiological factor syphilis or tuberculosis. Doubtful cases of this character should be subjected to a thorough course of antisyphilitic treatment.

# SECTION XIII

## *DISEASES OF THE SKIN*

## CHAPTER LXXXIV

### ECZEMA AND OTHER SKIN DISEASES

### ECZEMA

This is an inflammation of the skin which may manifest itself in an acute, subacute, or chronic form.

**Etiology.**—It is more common in infancy, since the very delicate structure of the skin at this period of life makes it especially susceptible to inflammation from irritants of any kind, and the unstable condition of the vasomotor nervous system makes it especially liable to cutaneous congestion, erythema, and inflammation from slight causes.

Any agent, chemical, toxic, infectious, physical or mechanical, which irritates the skin and is sufficiently intense and prolonged, will cause eczema. It matters not whether this irritation comes from without or from within.

Internal Causes.—Hereditary influences are important, since it is not uncommon to observe this disease in all of the children of a family, but the exact character of this hereditary predisposition cannot always be determined, but that in many instances it may be gouty or neurotic is beyond question. General malnutrition from such constitutional disturbances as glandular tuberculosis, syphilis, rickets, and anemia may produce special susceptibility to eczema. It is also stated that such reflex factors as diseased adenoids, adherent prepuce and dentition may be exciting factors; the importance of reflex factors, however, has been greatly exaggerated, and I have never been fully convinced that they deserve a place among the causative factors of this disease. The most important internal causes of eczema are undoubtedly autointoxications and gastrointestinal toxemias. Constipation, gastrointestinal indigestion, and food idiosyncrasies are important factors in many cases. It may be a very difficult matter in an individual case to determine the food idiosyncrasy which is producing the eczema. An article of food which in one child may aggravate or even produce an eczema will in another have no such influence. It is a fact, however, that overfeeding is one of the common factors in producing this disease.

External Causes.—Among the most important of these the following may be noted: Irritating soaps, rough handling of the tender skin, perspiration, the rubbing together of juxtaposed skin surfaces, acid urine, chafing discharges from the intestines and other mucous membranes, irritating clothing, exposure to extremes of heat and cold, the application of certain drugs and chemicals, the scratching associated with parasitic skin diseases, and other causes that produce itching, and the inoculation of the skin with pathogenic bacteria.

The character of the eczema depends upon the severity of the exciting cause producing the irritation, and its course upon the length of time that this cause is active.

**Symptomatology.**—Inflammation of the skin is a very common disorder of childhood. It usually begins as a congestion or erythema, and acute inflammatory changes follow, which may produce a variety of lesions. These acute processes may be erythematous, papular, vesicular, or pustular. These various forms may remain distinct, or they may be different stages of the same attack of eczema, or, in other cases, the various skin lesions may be more or less commingled, or at least may be present in different parts of the body at the same time.

Erythema is very frequently found where opposed surfaces rub one another, such as the groins, the neck, and the axilla; it is also very common on the face. It is characterized by redness, swelling, and infiltration of the skin, and later one of the typical forms of eczema may develop.

Papular eczema is characterized by the presence of small red papules, which have a tendency to group themselves on the face and on the arms and legs. The surrounding skin is congested and the papules have a shot-like feel; as they coalesce they may produce a decided thickening of the skin, characterized by marked redness and extreme irritation.

The vesicular type is characterized by the formation of large numbers of small vesicles, which may coalesce and produce large vesicles filled with a thick serum, which, as the vesicles break, is poured out and dries upon the surface. On the scalp this condition is known as seborrhea, and the whole or a portion of the hairy scalp of the infant is covered with a yellow or brownish scab, which is composed of the dried, yellowish exudate, cast-off epithelium and hair. When this dried, scabby material is removed it leaves a red, raw, and weeping surface, which, after a time, is again covered with the same scabby exudate. A vesicular eczema of the face and of the arms and legs, which are also favorable sites for this disease in childhood, is of the weeping variety, and portions of the surface may be covered by a thin crust which is easily removed.

The pustular form is characterized by the presence of pustules; it may be an advanced stage of the vesicular variety. In this condition the pus-forming organisms play an important part in the pathological process. It may be one of the stages of seborrhea of the scalp above mentioned, and occurs most commonly on the hairy scalp and on the face.

Following the acute stages above described a variety of forms may re-

sult. These, however, do not represent any particular change in the pathological process, and therefore require no detailed description. Eczema crustosum refers to the formation of crusts such as have been already described. Eczema squamosum refers to a subacute variety of eczema in which there is a scaly formation. Eczema rubrum refers to the condition of the skin which occurs in vesicular and pustular eczemas, in which, with the removal of the crusts or scales, the superficial layers of the epithelium of the skin are also removed, and the lower and red layers are uncovered, leaving a raw, weeping surface.

In all forms of eczema the inflammatory process in the skin is accompanied by itching and burning. This symptom, while present in every form of eczema, is more aggravated in the papular form. It causes the child to scratch and rub the inflamed parts and thereby adds to the inflammatory process, and very materially interferes with the curative treatment of this disease.

Eczema, as a rule, produces no constitutional disturbance unless the greater portion of the body of the child is involved. In most cases the child is well nourished and continues to gain in weight and develop along normal lines. In those instances where constitutional, nervous, or gastrointestinal disorders are associated with eczema, these conditions are a cause rather than an effect of the eczema.

**Diagnosis.**—Eczema is to be differentiated from other inflammations of the skin. The greatest difficulty perhaps arises in differentiating papular and pustular syphilides from eruptions of the same character produced by eczema. In syphilis the skin eruption is more general, less acute, is associated with little or no itching, and other evidences of syphilis are commonly present to assist in the differentiation. Neglected scabies may produce a well-marked eczema. The differentiation of scabies associated with eczema can, as a rule, be made by the presence of the initial burrows and by the clinical history of the condition which corresponds in its onset to that of scabies. The contagious character of scabies and the presence of more than one case in a family may also assist in the differentiation.

**Prognosis.**—The prognosis of acute eczema in infancy and childhood is, as a rule, good in those cases in which the physician has the coöperation of a conscientious mother or nurse who will faithfully carry out every detail of treatment. The prognosis, however, in subacute and chronic cases, especially if they be associated with some nutritional fault or chronic intestinal disturbance, is not so good. Many of these cases, in spite of conscientious treatment, may continue for many months or even years. In these cases the disease may be greatly improved for a time, only to have the symptoms recur from undiscoverable causes.

**Treatment.**—General Treatment.—In undertaking the treatment of a case of eczema, the mother or nurse must be made to understand the importance of the general as well as the local treatment. A careful family and previous personal history of the child must be obtained, in order to ascertain whether there is any constitutional taint, hereditary idiosyn-

crasy, or local constitutional disorder on the part of the infant itself which may influence the eczema.

If the infant be sturdy, fat, and well developed, a gouty or uric acid diathesis, if it can be established, may furnish a clue to the constitutional treatment. In such cases it is of the utmost importance that the infant should not be overfed. Its food should not contain more calories than necessary to maintain its nutrition. It is also of the greatest importance that constipation, if it exists, should be carefully overcome. This may be done by adding to the child's milk a sufficient quantity of saturated solution of phosphate of soda, one-half tablespoonful perhaps, to each bottle or glass of milk. Milk of magnesia may be used for the same purpose. Alkalies, such as bicarbonate of soda and lime water, are also of value. Not only the quantity, but the character, of the food must be carefully scrutinized. It is advisable to exclude all raw fruit, such as orange juice, and many of these cases are materially benefited by diminishing the quantity of sugar taken. A sugar-free diet will, in some instances, materially assist in bringing about a cure. Food rich in sugar, such as stewed prunes, is contraindicated, and it is also advisable to use as little sugar as possible with the cereal foods which the child may be taking. The "ready-to-serve" cereals, which are now so extensively used because of their judicious advertisement, are contraindicated.

If the infant is suffering from some pronounced malnutrition, every attention must be given to ascertaining the cause of this constitutional disturbance and correcting it. If the child be rachitic or tuberculous, fresh air, cod-liver oil, and a properly selected diet are necessary to its cure. In every case of eczema it is absolutely necessary to give careful attention to the gastrointestinal canal. If anemia be present, some of the malt and organic iron preparations may be of value. In addition to these general rules it is of great importance in subacute and chronic cases to study the food idiosyncrasies of the individual child. Some clew to this may be had by inquiring into the food idiosyncrasies of other members of the family, and then again it may be discovered that certain foods, such as oatmeal, potatoes, eggs, and sweets, are followed by relapses; when this is the case such foods are to be withheld until it be definitely proven that they do no harm. If eczema occurs in a breast-fed baby, the breast-milk should be analyzed, and if it be found to contain an excessive quantity of fat or protein, the diet, exercise, and general hygiene of the wet-nurse should be carefully regulated along the lines outlined in the chapter on Breast-Feeding. The breast-milk in some instances may be so modified as to materially influence the course of the eczema, but one is never justified in weaning an infant, if it be developing along normal lines, simply to cure its eczema.

In cases that fail to yield to ordinary methods of treatment, such reflex causes of nervousness as eye-strain, adenoids and phimosis should be sought for, and if found should be removed by appropriate treatment. There can be no doubt but that vasomotor disturbances may aggravate and

prolong an eczema; it is therefore possible that reflex factors such as those above named may act in this way.

In the treatment of eczema it is of the greatest importance that the inflamed skin should not be irritated by frequent washings with soap and water, and above all that the child should be prevented from scratching and rubbing the inflamed areas. This is perhaps the most difficult part of the treatment; the pruritus is so intense in many of these cases that, unless the child be constantly watched, it will manage in some manner to temporarily relieve the itching by rubbing the inflamed parts against the bed-clothing, or against some portion of its body. To prevent this in eczema of the face and scalp, masks and bandages are so applied as to cover the whole inflamed surface, and at the same time the arms are incased in stiff sleeves of wicker or pasteboard, so that the child cannot reach the inflamed parts with its hands. Such devices in an individual case must be left to the ingenuity of the physician, it being understood that an important part of the treatment is the prevention of the scratching and rubbing of the inflamed skin.

Local Applications.—Mild inflammations of an erythematous or papulo-erythematous type may be successfully treated by dusting powders, soothing lotions, compresses, and sedative ointments. Cases of the papulovesicular type of a severe and acute character should be treated with ice-cold compresses of weak, astringent, or boric acid solutions.

Cases of the vesicular type, in which the vesicles have ruptured and the surface is oozing, should be treated with wet compresses or with ointments which contain a sufficient quantity of starch or zinc oxid to absorb the secretion and prevent crust formation. When the inflamed skin is covered with crusts, these should always be softened with olive oil and gently removed. This is of special importance when the hairy scalp is involved. Long-standing cases in which there is more or less infiltration of the skin should be treated by stimulating ointments or lotions containing salicylic acid, resorcin and tar. Itching should be combated by antipruritic lotions.

Erythema intertrigo and simple forms of acute eczema, not located on the hairy parts of the body, may be successfully treated by dusting powders of stearate of zinc, or of equal parts of oxid of zinc and starch. In these cases Starten's lotion is of great value.

℞ Zinci oxidi ..... ℥ ss
Pulv. calaminæ præp. ..... ℈ iv
Glycerini ..... ℥ i
Liquoris calcis ad. ..... ℥ vi

S. Shake and apply freely every three or four hours to the inflamed skin.

Another valuable sedative and antipruritic lotion recommended by Heidingsfeld is as follows:

℞ Potass. sulphuret. ..... ʒ ss
Zinci sulphat. ..... ʒ ss
Zinci oxidi ..... ʒ v
Aquæ calcis ..... ʒ vii ss
Aquæ dest. q. s. td. ..... ℥ vi

This preparation should be shaken and applied locally every four hours by means of an ordinary bristle brush.

These lotions are not only sedative, but on drying they leave a coating of oxid of zinc over the inflamed parts.

Ointments, on the whole, are of more value than other applications in the treatment of eczema; to obtain good results, however, it is of the utmost importance that they should be properly made, and that the materials of which they are composed, especially the bases, should be fresh; if they are prepared with decomposing bases, containing fatty acids and other irritating materials, they may do more harm than good. The milder ointments used in the treatment of acute eczema should be applied on strips of lint or other soft material to the inflamed skin, and held there by appropriate bandages. In eczema of the face and scalp a well-fitting mask for the face, and hood for the scalp, best serve the purpose of holding the ointment in position and preventing irritation of the part by scratching and rubbing. In the chronic and subacute forms of eczema, in which stronger and stimulating ointments may be required, it is best to make the application by lightly rubbing the unguent into the inflamed part; the degree of reaction which follows such an application will determine whether a stronger ointment is to be used; if the reaction is marked and the inflammation of the skin is increased by such an application, then the milder ointments suitable for the treatment of acute eczema are to be used for allaying this irritation. Hardaway and Grindon say: "As a routine prescription in almost all types of eczema in children, especially eczema rubrum, the following prescription has the widest range of usefulness:

℞ Zinci oxidi ..... ʒ i
Pulv. amyli ..... ʒ ii
Ung. picis liq. ..... ʒ ii
Ung. vaselini plumb. q. s. ad. ..... ℥ i

"In warm weather the amount of starch may be increased. In place of the tar, 2 or 3 minims of carbolic acid may be added to each ounce; and, instead of the oxid of zinc, an equivalent quantity of boric acid. In most cases, however, the formula as given is the best."

In beginning the treatment a sedative ointment should be prescribed:

℞ Bismuth subnit. ..... ʒ ii
Zinci oxidi ..... ʒ ss
Glycerini ..... ʒ iss
Acidi carbolici ..... ♏ xx
Vaselini ad. ..... ℥ ii

Hebra's formula is an excellent one in some cases:

℞ Emplastri diachyli ..... ℥ i
Vaselini ..... ℥ i

Another formula of equally wide range of usefulness, very generally and almost universally employed in all types of eczema where there is a tendency toward vesiculation or oozing, is the well-known salicylated paste of Lassar:

℞ Acidi salicylici ..... grs.xxx
Zinci oxidi ..... ʒ vi
Starch ..... ʒ vi
Vaselini ..... ℥ ii

In cases where there is a suspected local infection of staphylococcic nature, either of etiologic or of incidental character, the sulphur salicylated paste of Lassar can be used with most gratifying results:

℞ Zinci oxidi ..... ʒ v
Starch ..... ʒ v
Sulphur ..... ʒ ii
Acidi salicylici ..... grs.xxx
Petrolatum ..... ʒ ii

In all cases where itching is a more or less intolerant feature, the following well-known lotion can be used in connection with other treatment:

℞ Liq. carbonis ..... ʒ i
Aquæ dest. ..... ℥ vi

Sig. Apply every few hours to allay itching.

In subacute eczema the following is used to remove the scales and stop the itching, and it may be followed by one of the milder ointments above given:

℞ Zinci oxidi ..... ʒ i
Ung. picis liquidæ ..... ʒ ii
Ung. aquæ rosæ ..... ʒ ii
Lanolini ..... ʒ iv

The following bland paste, which dries and is easily washed off, may also be recommended:

℞ Zinci oxidi ..... ʒ ii
Pulv. amyli ..... ʒ ii
Acidi salicylici ..... grs. xv
Ung. aquæ rosæ ..... ℥ i

In the treatment of seborrhea of the scalp the crust is to be thoroughly moistened with olive oil, which may be applied on strips of lint which are held in position by a skull cap. After twelve or twenty-four hours of such an application, the crust is to be carefully removed without tearing or irritating the inflamed skin, and the following ointment is then to be applied:

| | | |
|---|---|---|
| ℞ | Acidi salicylici | ℈ i |
| | Sulphuris præcip. | ʒ ss |
| | Ung. aquæ rosæ q-s | ℥ i |

## URTICARIA

Urticaria is a vasomotor neurosis, characterized by intense pruritus and presenting a more or less characteristic skin eruption. It is commonly called hives or nettlerash.

**Etiology.**—The important predisposing etiological factor is an exaggerated instability or excitability of the vasomotor nervous system, which creates in the individual a special susceptibility on the part of this system to respond to the reflex, toxic and other exciting factors of this disease. These factors by their action on the susceptible vasomotor nervous system produce a localized congestion of the part affected, associated with an edema due to serous exudation. The excitability of the vasomotor nervous system may be hereditary, as urticaria is not infrequently a family affection, or it may be acquired as the result of disease, improper feeding, or bad hygienic surroundings.

Urticaria in childhood is very commonly associated with gastrointestinal disturbances, and especially with acid fermentations from overfeeding with fats, starches and sugars. Certain articles of diet, such as strawberries, acid fruits, shell-fish, oatmeal, and preserved meats, may precipitate an attack without producing any apparent gastrointestinal disturbance. Intestinal worms, undigested food, reflex excitants, insect bites, and injury to the skin may be exciting causes. Diphtheria antitoxins and other serums may be followed by an attack of urticaria.

**Symptomatology.**—Intense pruritus occurring suddenly without apparent local cause on the part of the skin is the most characteristic symptom. In a severe attack of acute urticaria the suffering produced by the intense itching may be almost unbearable; in milder cases, and fortunately these are much more common, the severe itching is confined to small portions of the body, and is of short duration.

The Eruption.—The most common urticarial eruption in childhood is composed of small red papules (urticaria papulosa), which may be widely scattered over the surface of the body or confined to one part. This eruption is associated with such intense pruritus that the skin is usually torn and injured by scratching; a complicating eczema may thus be produced. In children the eruption may also present the appearance, as it so commonly does in the adult, of wheels with red circumferences and

paler centers. These wheels are elevated and the indurated and edematous thickening can be felt with the finger. This is the ordinary nettlerash. This character of eruption may be so grouped as to produce a general redness and swelling extending over a large portion of the body; when the face is involved the features may be distorted and the eyes closed. Rarely, urticaria is associated with a formation of vesicles, and hemorrhages may occur in the wheels or papules. The pruritus and eruption may disappear from one part of the body to reappear at once in another. Even in acute attacks the symptoms may abate and be exacerbated from time to time over a period of days or weeks, but in the great majority of cases the attack is of much shorter duration. In the subacute and chronic forms of this disease the attacks may continue for weeks at a time, and may recur at frequent intervals from slight or undiscoverable causes.

Urticaria of Mucous Membranes.—If the respiratory mucous membranes are attacked, coryza, sibilant bronchitis, severe dyspnea, or violent asthma may result. If the gastrointestinal mucous membrane is affected a colliquative diarrhea may occur, continue for a number of hours, and then subside without medical treatment. These syndromes on the part of the respiratory and gastrointestinal mucous membranes may be followed or preceded by attacks of urticaria of the skin.

**Prognosis.**—In the acute forms the prognosis is good, inasmuch as the cause can usually be located and the symptoms readily controlled. Chronic urticaria may persist for years, resisting the most careful treatment.

**Treatment.**—This should be begun by a dose of castor oil or some saline cathartic. Following this, bicarbonate of soda should be given internally and carbolic acid in some form applied to the skin to relieve the itching.

℞ Acidi carbolici ..... ʒ i
Zinci oxidi ..... ʒ ss
Pulv. calaminæ præp. ..... ℈ iv
Glycerini ..... ʒ i
Liquor calcis ..... ʒ vii

The above prescription, together with the one which follows, is recommended by Hardaway and Grindon to relieve the pruritus of urticaria:

℞ Mentholis ..... 3 ii
Alcoholis ..... q. s.
Acidi carbolici ..... 3 ss
Lotionis zinci oxidi comp. ..... ʒ vi

These lotions are to be applied freely with a soft rag.

Bromid of potash, phenacetin, or antipyrin, in proper doses, may relieve the general nervous irritability and add much to the comfort of the patient.

The above treatment applies to the relief of the immediate attack, but the most important part of the treatment is yet to follow, and that

pertains not alone to the satisfactory convalescence from the acute attack, but to the prevention of subsequent attacks. As a guide to this end the individual case should be carefully studied to discover the exciting causes of the attack and the food idiosyncrasies of the patient. If no cause other than general nervous irritability associated with some malnutrition is discovered, then the treatment should be fresh air, a carefully selected diet, outdoor exercise, quiet surroundings, and such tonics as the individual case may demand. These may include cod-liver oil, iron, or arsenic. In recurring urticaria it is most important that the patient should be treated for the underlying neurosis. In the great majority of cases, however, one is able to find some acute gastrointestinal disturbance, or some food idiosyncrasy, which has acted as the exciting cause. Following the preliminary cathartic, gastrointestinal disturbances are to be treated by a diet carefully selected to come within the range of the physiological digestive capacity of the individual child. As a matter of routine, oatmeal, orange juice, raw fruits of all kinds, shell-fish, fish, pastry, and sweets are to be carefully avoided. In the treatment of chronic and subacute cases it is of importance to remember that some of these cases respond to a sugar or fat-free diet, making a satisfactory recovery when the cream is taken off the milk and saccharin is used instead of sugar. It has been my custom in troublesome cases to place the child upon skimmed milk. If the urticaria is controlled by this diet, then from time to time other articles of food are carefully added, until following the giving of some article of food an attack of urticaria is produced. Only by some such careful method as this can the physician arrive at the particular food idiosyncrasy which may be an important factor in continuing this troublesome disease.

Certain intestinal antiseptics, such as salol, wintergreen sodium salicylate, and carbonate of guaiacol, may be of value in those cases which are produced by gastrointestinal toxemia.

## FURUNCULOSIS

**Symptomatology.**—This is an infectious condition characterized by multiple superficial abscesses of the skin involving the sebaceous glands and spreading to the cellular tissue. It occurs most commonly, and the abscesses are much more severe and widespread, in malnourished infants than in those of good physique. In normal well-developed infants with better powers of resistance the abscesses are smaller, fewer, and much more likely to be confined to the sebaceous glands. In malnourished infants the cellular tissue and deeper layers of the skin are involved, and the disease is much more widespread and yields much less readily to treatment. In the milder cases staphylococci, especially the staphylococcus pyogenes aureus, are the infecting organisms. In the more severe cases streptococci take part in the destructive process. The furuncles are commonly located on the scalp, forehead and neck, and in the severe cases may be widely disseminated over

the body. They may vary from a pea to a walnut in size, and may be very superficial, or may penetrate beneath the deep layers of the skin. The affection is not characterized by constitutional symptoms, except in rare instances, where it results in a general sepsis. The anemia, malnutrition, and general feebleness of constitution which are present in the worst cases are predisposing causes rather than symptoms of this disease.

**Prognosis.**—This is good, except in those rare cases where a general sepsis follows.

**Treatment.**—The underlying constitutional condition in malnourished children requires fresh air, careful feeding, and appropriate tonics, such as cod-liver oil and the malt and iron preparations. Calcium sulphid in ¼- to ½-grain doses, three times a day, is very generally recommended. The local treatment consists in thoroughly cleansing the skin with a warm bath of soap and water, and then incising the abscesses. In malnourished children having many large abscesses it is better not to open all of these at once. The abscesses are to be carefully drained, and a second general bath is to be given for the purpose of cleansing the skin. Following this, the wounds are to be dressed with a moist antiseptic solution, such as 1 to 10,000 bichlorid of mercury. A mild sulphur ointment, 10 or 15 grains to the ounce of lanolin, is a valuable remedy in the after-treatment of these abscesses, or Vleminckx solution may be used:

℞ Calcis ..... ʒ iv ss
Sulphur floris ..... ʒ ix
Aquæ ..... ℥ xv
M. Boil to one-half pint, let stand 24 hours. Filter.
Sig. Paint locally with bristle brush once or twice a week.

If the skin of the child is kept clean and proper attention is given to its general health, this treatment will in a short time result in permanent recovery. In the few cases, however, which resist this treatment "staphylococcus vaccine" may be used. The vaccine treatment of chronic furunculosis is followed by very good results. The autogenous vaccines are to be preferred in all cases, but where these cannot be had "stock vaccines" may be used as recommended under Vaccine Therapy.

## ERYTHEMA MULTIFORME

This is an acute inflammatory disease of the skin, the etiology of which is not definitely known. It is, however, believed to be infectious. In many cases it is apparently closely related to, or caused by a, gastrointestinal toxemia. Some observers believe that the disease is at times a rheumatic manifestation. It is most frequently seen during the winter and spring months.

**Symptomatology.**—An erythema appears on the extensor surfaces of the hands and feet, and gradually spreads upward over the arms and legs, and may finally extend very widely over all parts of the body. In the

beginning the eruption is pink or light red in color, and gradually becomes a darker red. As a rule it spreads by appearing in spots along the line of the lymph vessels; these red spots gradually increase in size and coalesce with neighboring spots, producing a more or less extensive erythematous patch, which commonly has less color in the center than it has at the periphery. Associated with these thickened erythematous macules there may be papules and vesicles. This multiform eruption of macules, papules and vesicles is characteristic of this disease. Patches of purpura are occasionally seen. There is a sense of discomfort with little or no itching, but with slight fever and occasionally tenderness about the joints. Endocarditis may occur, but is an infrequent complication. Systolic murmurs may be heard at the apex of the heart.

Gastrointestinal symptoms resulting from acute indigestion and intestinal toxemia are not infrequently associated with this condition. It must be remembered, however, that there is a form of simple erythema due to gastrointestinal toxemia, which is quite distinct from the condition here described.

**Prognosis.**—This is favorable. The disease runs a benign course, terminating in recovery.

**Treatment.**—If gastrointestinal disorders be present, they should be treated by proper diet and medicines. But even in those cases in which there is no evidence of intestinal disorder, the treatment should be begun by clearing the intestinal canal with calomel, followed by castor oil or a saline laxative. The child is then to be placed upon a milk and cereal diet, and given some preparation of salicylic acid, such as aspirin, salol, or wintergreen sodium salicylate. In some instances there is little doubt but that these remedies exercise a favorable influence upon the course of this disease. Sedative ointments containing 1 drachm of bismuth or 15 grains of oxid of zinc to the ounce of lanolin, should be used to relieve the irritation of the skin; if itching be present, 10 minims of carbolic acid may be added.

## CONGENITAL ICHTHYOSIS

The etiology of this condition is unknown. It is congenital and hereditary. It occurs as a family disease, and may reappear through many generations.

**Symptomatology.**—This rather rare condition makes its appearance in early infancy, and when fully developed the skin presents a very characteristic appearance. It is dry, thickened, and covered with fish-like scales. These horny, closely adherent flakes are hard and brittle, and cannot, as a rule, be removed without causing pain. The dried skin is broken and fissured, especially where there are folds, as in the flexures of the joints. This eruption is most characteristic on the body and on the extensor surfaces of the extremities.

**Prognosis.**—This is unfavorable; there is no curative treatment for this disease.

**Treatment.**—The palliative treatment consists in softening and removing the scales by sulphur and alkaline baths. Following this the irritation of the skin may be relieved by mild sulphur or salicylic acid ointments, 10 or 15 grains to the ounce. Mild sedative antiseptic ointments may be used for softening and increasing the flexibility of the skin.

## IMPETIGO CONTAGIOSA

**Etiology.**—This is an inflammatory condition of the superficial layers of the skin produced by microorganisms. Streptococci and staphylococci are found, but the specific organism of this affection is not at the present time definitely known. It is a distinctly contagious disease, characterized by definite lesions. It can be transmitted by inoculation from one individual to another, and is readily transferred from one part of the body to another by scratching. The fact, however, that such inoculations produce the same characteristic lesions is proof of the specificity of the microorganism which causes the disease. It may occur at all ages, but it is much more common in children than in adults.

FIG. 105.—IMPETIGO CONTAGIOSA. (M. Heidingsfeld.)

**Symptomatology.**—The primary eruption occurs in the form of thin watery vesicles, which in a short time are filled with seropurulent or purulent fluid. These pustules break and the exuded matter dries and produces a yellow scab, which is attached very loosely to the surface, and is surrounded by little or no redness of the skin. Crops of vesicles spring up around this scab, run the same course, and, as a result, a large portion of the face, especially about the angles of the mouth and chin, is covered with these yellow crusts. As the scab drops off, or when it is removed, the underlying skin is red and presents a raw, moist appearance. The eruption occurs most commonly on the lower portion of the face, and may be transferred from there by inoculation to other portions of the face, to the hairy scalp, and to the hands. These uncovered parts are most likely to be infected, but other parts of the body may also be inoculated, so that the disease may be very widespread. At times the scabbing of the skin as the lesion becomes older may present circular or oval forms, the centers of these patches having healed. It is scarcely possible to mistake this form of impetigo for ordinary eczema. The manner of its spread and the uni-

formity with which it presents the vesicle, pustule and scab should make the differentiation plain.

**Treatment.**—The treatment of this condition is simple and very satisfactory. It yields very readily to mild sulphur and salicylate ointments. In beginning the treatment the crust should be softened with oil, lanolin, or vaselin, and then carefully removed. Sulphur soap may be used for the washing of the diseased part.

Following the removal of the scab one of the following ointments may be applied:

℞ Acidi salicylici ..... grs. xv
Zinci oxidi ..... grs. xv
Vaselini ..... ℥ i

℞ Hydrargyri ammoniati ..... grs. vii
Vaselini ..... ʒ iv
Lanolini ..... ʒ iv
Sig. Apply on lint.

## PEMPHIGUS NEONATORUM

This is an infectious disease of the skin which makes its appearance soon after birth. It is characterized by the appearance, between the third and the tenth day of life, of large vesicles or cysts varying from 1/8 to 1 inch in size. These vesicles are filled with a cloudy serum, and the surrounding skin is usually slightly reddened. As the thin vesicles break the serum dries and forms a thin crust, which, on removal, leaves a reddened raw surface. This eruption usually appears over the whole body, with the exception of the palms of the hands and the soles of the feet. New vesicles appear from time to time, the disease running its course in five or six weeks. In the great majority of cases it is benign, has no constitutional symptoms, and terminates in complete recovery. Occasionally, however, the disease may manifest itself in a more malignant type, being associated with symptoms of general sepsis. These cases are prolonged, and, as a rule, terminate fatally.

**Etiology.**—It is believed that pemphigus neonatorum and impetigo contagiosa are produced by the same contagion, the difference in the appearance of the eruption depending upon the age of the child. In the newly born infant it presents the appearance of pemphigus just described, while in the older infant and child the clinical symptoms of impetigo are produced. Staphylococci and streptococci found in the two diseases are similar, and inoculation experiments indicate the identity of the two conditions. The specific microorganism, however, of this infection has not been clearly demonstrated.

**Diagnosis.**—The characteristic bullous eruption of this disease, occurring as it does only during the early days of life, can be mistaken only for syphilitic pemphigus. In this latter condition, however, the blebs

are most commonly found on the palms of the hands and the soles of the feet. This fact, with other symptoms of syphilis, which can always be found, is sufficient to make the differentiation.

**Treatment.**—The great majority of the cases require little treatment. The skin eruptions may be treated with a dusting powder of stearate of zinc or mild antiseptic ointments, such as the following:

| | | |
|---|---|---|
| ℞ | Bismuth subnit. | ʒ i |
| | Acidi borici | ʒ ss |
| | Lanolini ad | ʒ ii |

Where the disease is associated with constitutional symptoms, the treatment is the same as that outlined for septic infection of the newborn.

## TINEA TONSURANS

Tinea tonsurans, or ringworm of the scalp, is a contagious disease caused by a vegetable parasite. As Saboraud demonstrated, this parasite occurs in a variety of species which produce slightly different clinical syndromes. This form of tinea is confined almost exclusively to children, and is usually spread by direct contact; it may, however, be conveyed through articles of clothing and toilet utensils. It is very frequently seen in institutions and families in epidemic form.

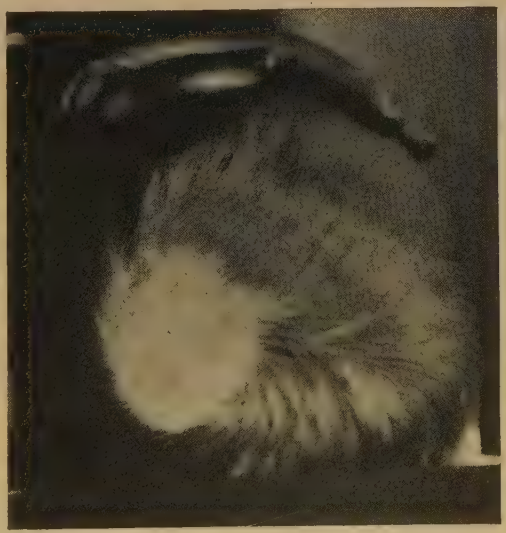


Fig. 106.—Alopecia Accompanying Ringworm of the Scalp. (M. Heidingsfeld.)

**Symptomatology.**—Ringworm of the scalp occurs in round or oval patches which are surrounded by a slightly raised and reddened ring. Toward the center of this ring the skin grows paler, and the hairs within the patch are stiff, brittle, broken off near the skin, and surrounded by a whitish scaly epithelium. The patch increases in size in all directions, gradually producing a larger ring, which approaches baldness. The same child may have more than one patch of tinea, and these, by enlarging, may run together, producing one large irregular patch with rounded ends.

**Diagnosis.**—The diagnosis is easily made by the presence of circular scaly patches having a reddened circumference and containing the dry and brittle hair stumps. If the roots of one of these diseased hairs be exam-

ined under the microscope, after soaking it on a cover glass in a drop of liquor potassii, the small spores which constitute this parasite may be discovered.

**Treatment.**—The treatment of this condition is entirely local, and, on the whole, is satisfactory, although many cases respond very slowly to treatment.

The prophylactic treatment is important. Children with tinea should not be allowed to go to school, and in institutions they should, if possible, be isolated from other children. Where isolation is not possible, every precaution should be taken against the spread of this disease by prohibiting diseased children from coming in close contact with the well ones, and by seeing that each child suffering from tinea has his own comb and hairbrush, and that these be frequently disinfected. All toilet articles and clothing used by infected children should be carefully boiled and washed before they are used by other children.

In beginning the treatment the hair of the entire head should be closely cut or shaven. In girls this is not always possible, as the physician is requested to make an attempt to save the hair; in such cases the hair for half an inch around the patch of tinea should be closely cut. After this preparation the whole scalp is to be carefully washed with soap and water, and afterward with a saturated solution of boracic acid. This washing of the scalp with boracic acid is to be done daily throughout the treatment, not only to prevent the spread of the disease to other children, but to prevent inoculation of other portions of the scalp in the same child.

Sulphur and salicylic acid are very valuable in the treatment of ringworm. These remedies may be combined in the same prescription as follows:

℞ Acidi salicylici ..... ʒ ss
Sulphuris præcip. ..... ʒ i
Vaselini ..... ℥ i
Sig. Rub thoroughly into the patch morning and evening.

This ointment is also of great value in ringworm of the body, which yields very readily to treatment.

Chrysarobin is one of the most valuable of remedies in ringworm of the scalp. It, however, must be applied carefully to the patch and subsequent applications are to be decided on by the degree of reaction which follows the first application. Sometimes it causes an acute eczema which contraindicates its use. Hutchinson, quoted by Hardaway and Grindon, recommends the following formula:

℞ Chrysarobini ..... ʒ i
Hydrarg. ammoniati ..... grs. xx
Liq. carbonis deterg. ..... ♏ x
Lanolini ..... ʒ i
Adipis recentis ..... ʒ vi

They also say the following method, recommended by Crocker, "has given good results in our hands." The patches, as well as a surrounding strip one-half inch wide, are closely shaven, after which they are painted with collodion containing salicylic acid, 1 to 30. Fresh collodion is applied every day for a week. The dried collodion is then lifted off by inserting a spatula under its edge, and the process repeated until a cure is effected.

Heidingsfeld says an almost unfailing chrysarobin ointment, which should be applied exceedingly sparingly, is the following:

| | | |
|---|---|---|
| ℞ | Acidi salicylici | grs. xxx |
| | Acidi pyrogallici | ʒ i ss |
| | Resorcini | ʒ i |
| | Chrysarobini | ʒ i ss |
| | Sapo viridis | ʒ v |
| | Petrolati | ʒ x |

Cases that do not yield satisfactorily to the above treatment should be treated by the X-ray; when this is properly done the disease commonly yields very readily. A permanent and comparatively rapid cure is effected.

## SCABIES

**Etiology.**—Scabies, or the itch, is a contagious disease of the skin caused by the acarus scabiei (itch-mite). This parasite burrows between the epithelial layers of the skin, producing a thin line from ⅛ to ½ inch in length, which can readily be seen through a small magnifying glass, and may be made out by the naked eye. This burrow is usually slightly discolored with dirt, especially at its entrance, and at its end a yellowish opaque object (the acarus) and a pearly white vesicle may be seen. The acarus may be lifted from its burrow on the point of a needle and examined under the microscope; the finding of the itch-mite is absolute proof of the existence of scabies. The diagnosis, however, is commonly made by the characteristic burrows above described and by the intense itching which occurs early, continues throughout the disease, and is always worse at night. These pathognomonic burrows can be most readily found between the fingers and upon the wrists, feet, and buttocks of the child. Very commonly there is difficulty in finding them because they are obscured by the secondary inflammatory lesions, produced in part by the irritation of these parasites, but largely by the scratching and tearing of the skin, which are unavoidable on account of the intolerable itching. In nearly all cases a secondary eczema occurs, characterized by the formation of vesicles, papules and pustules. These are lacerated by scratching, and finger-nail marks are added to the other lesions, and the serum and pus dry, producing crusts.

**Diagnosis.**—In advanced cases this disease may be mistaken for ordinary eczema. When the burrows can be seen and the parasites found the

diagnosis is easy. But even when this is not possible the differential diagnosis between the two conditions can usually be made by the intense pruritus which characterizes scabies from the very beginning of the disease, and by the favorite locations of the two conditions. A history pointing to contagion is also important.

Fig. 107.—Pustular Scabies of the Hands. (M. Heidingsfeld.)

**Treatment.**—The treatment is simple and very satisfactory; sulphur applied in the form of an ointment acts specifically in the cure of this disease. Before making this application all personal and bed-clothing should be sterilized to prevent reinoculation during or following the treatment. The whole surface of the body should be cleansed with soap and hot water and, after thoroughly drying the skin, all that portion of the body on which there is any eruption should be carefully rubbed with sulphur ointment—1 drachm of sulphur to 1 ounce of vaselin. This application should be made before going to bed, and the next morning it may be removed by a bath of soap and hot water, and clean clothes put on for the day. This treatment is to be repeated three or four nights in succession. It may then be discontinued for a few days, and again repeated for two nights in succession; by this time a permanent cure is usually effected so far as the destruction of the acaries is concerned, but the associated eczema may require treatment.

## PEDICULOSIS CAPILLITII

**Etiology.**—This condition is caused by the pediculus capitis, or head louse, which is 1 to 2 mm. in length and has attached to its head a sharp proboscis through which it feeds by imbedding it in the scalp. This produces the intense itching which is characteristic of this condition, and causes the child to scratch, tear, and mutilate the already irritated scalp. The irritation caused by the louse, together with the traumatism produced by the scratching, may cause an inflammation of the skin (eczema), which is confined to the hairy scalp. This parasite is very prolific and fastens

its eggs in great numbers to the hairs; these may be readily seen as grayish-white scales rather firmly attached to the hair. They may be distinguished from dandruff scales by the fact that they cannot be removed by brushing.

The eczema produced by this condition is characterized by much more itching than is ordinary eczema of the scalp. The lymphatic glands in the occipital region are enlarged in aggravated cases.

**Treatment.**—In hospital cases the hair should be closely cut; in private practice, however, the condition is usually discovered before a well-pronounced eczema has resulted, and the cutting of the hair is not necessary.

The treatment consists in saturating the hair with coal-oil. This may be done by carefully rubbing it in with a cloth. When the hair is thoroughly soaked, a skull cap is applied covering the whole hairy scalp; the next morning the oil is washed out with soap and water and the hair dried. If the associated eczema persists, it may require treatment, but, as a rule, with the removal of the cause the inflammation of the skin is quickly cured.

Heidingsfeld says: "It is essential for the successful treatment of pediculosis capillitii that one should bear in mind that the developing larvæ are not destroyed by the antiseptics which are successful in removing insects which have been hatched. The larvæ are encased in impervious keratin, and, after an interval of seven to fourteen days, there will be a fresh crop of new insects. These must be destroyed by a repetition of the treatment. The larvæ are attached to the hair by means of a calcareous cement. This is readily dissolved by ordinary vinegar or a ten per cent. solution of acetic acid. The live insects and the larvæ, however, can be removed at the same time by saturating the hair with compresses of 1-200 bichlorid in vinegar, for six or eight hours. The bichlorid destroys the live insects and the larvæ are loosened from the hair by the vinegar, so they can be readily removed by means of a fine comb."

# INDEX


ABDOMEN, examination of, in illness, 30.

ABSCESS, of brain, 617; deep bone, complicating typhoid fever, 274; of liver, complicating lobar pneumonia, 448; of lymph glands, complicating vaccinia, 353; periesophageal, 157; perinephritic, 590; peritonsillar, 421; retropharyngeal, 427.

ABT, on renal and vesical irritation following use of urotropin, 587.

ACARUS SCABIEI, 735.

ACETATE OF POTASH. *See Potassium acetate.*

ACETONE GROUP, presence of, in urine, 565; in recurrent vomiting, 255.

ACETONURIA, 565; diagnosis of, 566; etiology of, 565; in measles, 336; prognosis of, 566; treatment of, 567; in typhoid fever, 273.

ACIDOSIS, in recurrent vomiting, 252, 255.

ADAMS, report by, of recoveries from septic endocarditis, 491.

ADÉNIE. *See Hodgkin's disease.*

ADENITIS, CERVICAL, in scarlet fever, 321; treatment of, 331.

ADENITIS, SIMPLE, 542; definition of, 542; diagnosis of, 544; etiology of, 542; microorganisms present in, 542; symptomatology of, 543; treatment of, 544.

ADENOIDS, 424; cough caused by, mistaken for whooping cough, 291; diagnosis of, 426; etiology of, 425; frequency of, 425; history of knowledge concerning, 424; method of examining, 426; as cause of pavor nocturnus, 672; sources of infection in, 425; symptomatology of, 425; treatment of, 426.

ADENOIDS as portals of infection, in endocarditis, 493; in acute articular rheumatism, 402; in chorea, 701; in diphtheria, 295; in influenza, 311; in status lymphaticus, 545, 547.

ADENOIDS, removal of, as protection against acute articular rheumatism, 406; for relief of asthma, 712; as protection against endocarditis, 493, 503; indications for, 427; as protection against pericarditis, 512; for relief of acute rhinitis, 414; for relief of chronic rhinitis, 415; for relief of tonsillar hypertrophy, 424; as protection against tuberculosis, 395.

ADRENALIN, use of, in asthmatic attacks, 712; in epistaxis, 416; in hemophilia, 539; in hemorrhage in new-born, 83; in purpura hemorrhagica, 537; in splanchnic paralysis during diphtheria, 307.

AGGLUTININS, 55.

ALBOLIN, use of, in scarlet fever, 330.

ALBUMIN WATER, as artificial food, 128; in infantile atrophy, 201; in typhoid fever, 278.

ALBUMINURIA, 567; cyclic, 568; definition of, 567; forms of, 567; in influenza, 312; in mumps, 359; in nephritis, 576; orthostatic, 568; physiological, 567; postural, 568; in purpura hemorrhagica, 533; in purpura rheumatica, 535; in scarlet fever, 322; in septic infection of new-born, 78; in tonsillitis, 422; in variola, 347; in whooping cough, 290.

ALBUMINURA, toxic. *See Nephritis, toxic.*

ALCOHOL, therapeutic use of, in bronchopneumonia, 466; in acute cardiac dilatation, 498; in lobar pneumonia, 450; multiple neuritis due to undue use of, 661; in toxic nephritis, 574; in typhoid fever, 277.

ALEXINS, 107.

ALKALIES, therapeutic use of in acetonuria, 567; in acute articular rheumatism, 405; in enuresis, 602; in pica, 705.

ALKALINE DILUENTS, 139.

ALKALINE WATERS, therapeutic use of, in cystopyelitis, 586; in eczema, 722; in acute nephritis, 581; in chronic nephritis, 583; in toxic nephritis, 574; in valvular heart disease from rheumatism, 504.

AMAUROTIC FAMILY IDIOCY, 624.

Ameba coli, 178.
Amyl nitrite, use of, in epilepsy, 695.
Amylase, in human milk, 106.
Anaphylaxis, 185.
Anatomic age of child, 19.
Anderson and Frost, on the extent of paralysis in acute anterior poliomyelitis, 647.
Anemia, simple, 522; due to acute articular rheumatism, 404, 406; blood picture in, 523; cause of chorea, 697; symptom of chorea, 700; definition of, 522; diagnosis of, 523; due to endocarditis, 490; etiology of, 522; associated with habit spasm, 702; associated with hemophilia, 539; associated with Hodgkin's disease, 541; due to malaria, 283; prognosis of, 524; due to purpura hemorrhagica, 533; associated with splenomegaly, 552; associated with sporadic cretinism, 554; symptomatology of, 523; due to syphilis, 366; associated with toxic nephritis, 572; treatment of, 524; associated with tuberculosis of lymph nodes, 382; associated with valvular heart disease, 505.
Anemia of infancy and childhood, 518, 521.
Anemia, pernicious, 528; blood picture in, 528; definition of, 528; diagnosis in, 529; etiology of, 528; symptomatology of, 528; treatment of, 529.
Anesthesia, symptom of hysteria, 706.
Anesthetics, danger from, in status lymphaticus, 550; sudden death from, 547.
Angina, gangrenous, in scarlet fever, 324; ulcerative, 324.
Angioneurotic edema, 535.
Anisocytosis, 516.
Anopheles, destruction of, 284; transmission of malaria by, 280.
Anorexia nervosa, in hysteria, 707.
Antibodies, syphilitic, 368.
Antimeningitic serum. *See Serum.*
Antipyretics, use of, in bronchopneumonia, 467; in fevers, 266; in lobar pneumonia, 451; in scarlet fever, 329; in typhoid fever, 278.
Antiserums, therapeutic use of, 59.
Antitoxin of diphtheria, use of, in bronchopneumonia complicating diphtheria, 307; use of, in determining diphtheritic character of pseudo-membranous rhinitis, 413; use of, in diagnosis between diphtheria and acute laryngitis, 432, 433; use of, in diagnosis between diphtheria and tonsillitis, 420; discovery of, 302; dose of, in hospital practice, 303; dose of, in private practice, 304; use of, with intubation, 304; use of, in membranous laryngitis complicating measles, 340; method of preparing, 302; method of using, 303; mortality of diphtheria reduced by, 300, 302; use of, in post-diphtheritic paralysis, 307; prophylactic use of, 302; use of, in scarlet fever complicating diphtheria, 330; use of, in septicemia complicating diphtheria, 307; skin eruptions following use of, 304; use of, in stomatitis gangrenosa, 153; use of, with tracheotomy, 304.
Antitoxin of tetanus, 59, 91.
Anti-typhoid inoculation, 276.
Anuria, 560.
Anus, atresia of, 233; fissure of, 235; malformation of, 233; spasm of, 236.
Aortic regurgitation, 501.
Aortic stenosis, 501.
Aphonia, hysterical, 707.
Appendicitis, 222; blood picture in, 225; conditions covered by term, 222; diagnosis of, 226; etiology of, 223; indications for operation in, 228; mortality from, 227; pathology of, 223; physical examination in, 224; prognosis of, 227; symptomatology of, 224; treatment of, 227.
Argyrol, local use of, in diphtheria, 304; in acute follicular tonsillitis, 423; in gonorrheal vulvovaginitis, 594; in scarlet fever, 330.
Arnheim, on Röntgen-ray picture in diagnosis of persistent patulous ductus arteriosus Botalli, 488.
Arnold steam-sterilizer, 120.
Arrhythmia, 506.
Arsenic, therapeutic use of, in asthma, 713; in acute cardiac dilatation, 498; in chorea, 700; in enuresis, 601; in Hodgkin's disease, 542; in malaria, 286; as cause of multiple neuritis, 661; in pavor nocturnus, 675; in pernicious anemia, 529; pseudo-leukemia unaffected by, 526; in pseudo-masturbation, 609; in simple secondary anemia, 524; toxic nephritis caused by, 573, 575; in valvular heart disease, 505.
Arsenic, Fowler's solution of. *See Fowler.*
Arthritis, chronic rheumatoid, 408.
Arthritis, chronic villous, 407.
Arthritis deformans. *See Chronic rheumatoid arthritis.*
Arthritis, infectious, 407.

Arthritis, septic, complicating scarlet-fever, 324.
Artificial feeding in infancy, additional foods in, 142; albumin water in, 128; alkaline diluents in, 139; beef juice in, 129; broths in, 129; buttermilk in, 122; caloric standard of values in, 130; cane sugar in, 125; carbohydrates in, 124, 135; carbohydrate diluents in, 138; casein in, 135; cereal decoctions in, 126; cleanliness essential in, 133; condensed milk in, 126; cow's milk in, 118; determination of accurate percentages in, 139; dextrinized gruels in, 126; fat in, 135; Finkelstein's albumin milk in, 123; food formulæ in, 133; fresh air essential to success of, 134; home modification of milk in, 135; ingredients of, 103; malted milk in, 128; maltose in, 125; malt soups in, 124; meat preparations in, 129; necessity for, among poor, 115; Nestlé's food in, 127; overfeeding a danger in, 133; pasteurized milk in, 120; peptonized milk in, 121; percentage of different foodstuffs in, 134; in premature infants, 67; principles of, 132; proprietary foods in, 127; regularity essential to success of, 134; rest essential to success of, 134; Rotch laboratory method of modification in, 141; skimmed milk in, 124; sterilized milk in, 119; value of percentage feeding in, 129; in treatment of whooping cough, 292.
Artificial respiration, 72.
Ascaris lumbricoides, 215; treatment of, 216.
Ascites, 232; diagnosis of, 232; etiology of, 232; treatment of, in tumor of the kidney, 589; treatment of, in valvular heart disease, 506.
Asphyxia, as cause of idiocy, 623; livida, 70; pallida, 70.
Asphyxia neonatorum, 69; diagnosis of, 71; etiology of, 69; prognosis of, 71; prophylaxis of, 71; symptomatology of, 70; treatment of, 72.
Aspidium, use of, in tenia, 214.
Aspiration, in pericarditis with effusion, 513; in pleurisy with effusion, 479.
Aspirin, therapeutic use of, in endocarditis, 494; in fevers, 267; in influenza, 314; in pleurisy, 478; in tonsillitis, 422; in tuberculosis, 400.
Astasia-abasia, 706.
Asthma, 710; definition of, 710; etiology of, 711; prognosis of, 712; symptomatology, 711; thymic, 546; treatment of, during attack, 712; treatment of, during interval, 712.
Ataxia, hereditary, 657; treatment of, 658.
Atelectasis, congenital, 73; etiology of, 74; symptomatology of, 74; treatment of, 75.
Atrophy, muscular, in facial paralysis, 665; in multiple neuritis, 663; in acute anterior poliomyelitis, 648; affecting the voluntary muscles, 666.
Atropin, therapeutic use of, in asthma, 712; in ophthalmia neonatorum, 97.
Aura, 693.
Auscultation, value of, in physical examination, 34.
Autogenous vaccines, therapeutic use of, 57.
Autoinfections, a cause of headache, 709; association of, with toxic nephritis, 573.
Autotoxins, cause of eczema, 719; cause of headache, 709.

B

Babinski reflex, in epilepsy, 694; in meningococcus meningitis, 633; method of examination for, 32; in muscular contracture of infantile cerebral palsy, 613; in tuberculous meningitis, 628.
Bacillus: B. "blue," cause of enteric infection, 178; B. coli communis, *see B. colon;* B. colon, cause of cystopyelitis, 584; cause of enuresis, 601; cause of fever resembling typhoid, 267; cause of simple vulvovaginitis, 595; B. enteriditis, cause of fever resembling typhoid, 265, 267; cause of meat-poisoning, 267; B. gas, *see B. Welchii;* B. of influenza, 308, 311; cause of enteric infection, 178; B. Klebs-Löffler, 295; B. paratyphoid, cause of fever resembling typhoid, 265, 267; cause of meat-poisoning, 267; B. pertussis, 287; B. Pfeiffer, *see B. of influenza;* B. proteus, as cause of enteric infection, 178; B. pseudo-diphtheritic, as cause of simple vulvovaginitis, 595; B. Shiga, 178; B. typhoid, 267; B. of Vincent, 419; B. Welchii, as cause of enteric infection, 178.
Bacteria, present in appendicitis, 223, 224; present in human milk, 112; present in intestinal canal of infant, 176, 177; entrance of, through umbilical wound in sepsis of new-born, 263.

Bacterial content, in cream, 133; in milk, 118, 119.
Babes and Zambolovici, bacillus isolated by, found in gangrenous stomatitis, 152.
Bacterial substances. *See Bacteriolysins.*
Bacterial vaccines, therapeutic use of, 55.
Bacteriological examination, in diagnosis between brain abscess and meningitis, 618; in diagnosis of diphtheria, 301; in prophylaxis of diphtheria, 302; in diagnosis between diphtheria and acute laryngitis, 432, 433; in diagnosis between diphtheria and tonsillitis, 420; in diagnosis of pleurisy with effusion, 477; in diagnosis of acute poliomyelitis, 649; in diagnosis of tubercular meningitis, 629.
Bacteriolysins, 55.
Baginsky, on frequency of nephritis as complication of diphtheria, 300.
Barlow, on acute articular rheumatism, 402; on acute articular rheumatism attacking the hip joint, 403; on infantile scurvy, 246.
Basophiles, 517.
Baths, alkaline, in treatment of epilepsy, 696; in treatment of purpura, 536.
Baths, cold, objections to, in acute catarrhal bronchitis, 442; objections to in typhoid fever, 277; in treatment of fever in infancy, 266; in treatment of fever in older children, 267.
Baths, hot, in edema of larynx, 435; in lobar pneumonia, 451; temperature of, 46; therapeutic value of, 46.
Baths, Nauheim. *See Nauheim.*
Baths, salt, in treatment of rickets, 245.
Baths, sponge, in bronchopneumonia, 468; in cystopyelitis, 587; in fever of infancy, 267; in lobar pneumonia, 451; in malaria, 286; in measles, 339; in scarlet fever, 329; in tonsillitis, 423; in variola, 348; temperature of, 45; therapeutic value of, 45.
Baths, tepid, in fever of infancy, 267; in influenza, 313; in typhoid fever, 277.
Baths, tub, in eclampsia, 682; in scarlet fever, 329; in tonsillitis, 423; objections to, in typhoid fever, 277; temperature of, 45; therapeutic value of, 45.
Baths, warm, in asphyxia neonatorum, 73; in bronchopneumonia, 468; in acute catarrhal bronchitis, 442; in chorea, 701; in acute laryngitis, 433; in meningococcus meningitis, 638; in multiple neuritis, 663; in tetany, 689; in toxic nephritis, 574; in tuberculous bronchopneumonia, 401.
Bäumler, on results of vaccination, 350.
Becamp, discovery of amylase by, 106.
Beck, on pneumococcus as cause of pleurisy in child, 470.
Bednar's aphthæ, 154.
Bedsores in myelitis, 654, 656.
Beef juice, value of, in artificial feeding, 129.
Behring, discovery of antitoxin by, 302.
Bell, on facial paralysis, 665.
Bell's palsy. *See Facial paralysis.*
Belladonna, therapeutic use of, in asthma, 712; in bronchopneumonia, 467; in acute catarrhal bronchitis, 441; in cough of tuberculosis, 400; in enuresis, 602; in epilepsy, 696; in influenza, 313, 314; in lobar pneumonia, 453; in measles, 339; in pavor nocturnus, 675; in pseudo-masturbation, 609; in whooping cough, 293.
Belladonna ointment, use of, in mumps, 360.
Beneke, on increase in size of the heart during first five years of life, 484.
Benoit, on digestive ferments present in human milk, 107.
Benzoate of soda. *See Sodium benzoate.*
Benzoin, compound tincture of, use of, by inhalations, in acute laryngitis, 433; in acute rhinitis, 414.
Bernheim and Pospischill, on stomatitis ulcerosa, 150.
Bernouilli, on mortality from small-pox before vaccination, 349.
Betanaphthol, in chlorosis, 528; in pernicious anemia, 529; in pseudo-leukemia, 526.
Bicarbonate of soda. *See Sodium bicarbonate.*
Bicarbonate of potash. *See Potassium bicarbonate.*
Bile ducts, occlusion of, in new-born, 93.
Birth injuries, 97; birth palsies, 98; cephalhematoma, 97; facial paralysis, 98; hematoma of the sterno-cleido-mastoid muscle, 98; upper-arm paralysis, 99.
Birth palsies, 98.
Bismuth subnitrate, external use of, in

erythema multiforme, 730; in syphilitic ulcerations, 373.
Bismuth subnitrate, internal use of, in diarrhea of tuberculosis, 400, 401; in diarrhea of typhoid fever, 278; in enteric infection, 195.
Blackader, on prognosis in typhoid fever, 275; on relapses in typhoid fever, 274.
"Black measles," 335.
Blaud's pills, therapeutic use of, in chlorosis, 528; in purpura, 536.
"Bleeders," 537.
Blisters, in treatment of facial paralysis, 666; in treatment of hysteria, 708; contraindicated in treatment of acute pericarditis, 512.
Blood, color index of, 516; diseases of, 515; diagnostic importance of, 37; peculiarities of, in infancy and childhood, 518; physiology of, 515; platelets of, 517; red corpuscles of, 515; white corpuscles of, 516.
Blood, character of, in acute articular rheumatism, 404; in appendicitis, 225; in chlorosis, 527; in chorea, 700; in congenital heart disease, 486; in diphtheria, 297; in Hodgkin's disease, 541; in influenza, 310; in leukemia, 530; in lobar pneumonia, 447; in malaria, 284; in measles, 336; in meningitis, 634; in pernicious anemia, 528; in poliomyelitis, 649; in pseudo-leukemia, 525; in purpura, 532; in recurrent vomiting, 254; in rickets, 242; in rubella, 343; in scarlet fever, 322; in simple secondary anemia, 523; in status lymphaticus, 547; in syphilis, 366, 368; in typhoid fever, 273; in varicella, 356; in variola, 347; in whooping cough, 289.
Blood corpuscles, red, function of, 515; number of, 515; origin of, 518; pathological changes in, 515; reduction of, in chlorosis, 526, 527; reduction of, in Hodgkin's disease, 541; reduction of, in leukemia, 530; reduction of, in pernicious anemia, 528; reduction of, in simple secondary anemia, 523; varieties of, 515.
Blood corpuscles, white, number of, 516; origin of, 516; pathological changes in, 517; varieties of, 516.
Blood, diseases of, 515; chlorosis, 526; hemophilia, 537; leukemia, 530; pernicious anemia, 528; pseudo-leukemia, 524; purpura, 531; simple secondary anemia, 522.
Blumenreich, on area of dulness produced by the thymus gland, 547.
Boggs, on changes in area of thymic dulness, 547.
Bokay, on frequency of retropharyngeal abscess in first year of life, 428.
Bones, changes in, due to syphilis, 365, 367; changes in, due to tuberculosis, 373, 381, 391; deformities of, due to rickets, 240; treatment of tuberculosis of, 401.
Bony framework, growth and development of, 19.
Booker, on proteus vulgaris as a cause of enteric infection, 178; on streptococcus enteriditis as a cause of enteric infection, 178.
Boracic acid, as irrigating solution in vulvovaginitis, 594.
Bordet and Genow, isolation of "bacillus pertussis" by, 287.
Bothriocephalus latus, 212.
Bovaird, on primary intestinal tuberculosis, 380.
Bowditch, on measurements of growth in children, 17.
Boyd, on weight of brain in early life, 23.
Bradycardia, 507.
Brain abscess, 617; course of, 618; diagnosis of, 618; duration of, 618; etiology of, 617; situation of, 617; symptomatology of, 618; treatment of, 618.
Brain, diseases of, 610; abscess, 617; amaurotic family idiocy, 624; encephalocele, 621; hydrencephalocele, 622; hydrocephalus, acute, 618; hydrocephalus, chronic, 618; idiocy, 622; meningocele, 621; microcephalic idiocy, 625; Mongolian idiocy, 624; tumors, 616.
Brain tumors, 616; diagnosis of, 617; general symptoms of, 616; local symptoms of, 617; nature of, 616; position of, 616; treatment of, 617.
Brandy, therapeutic use of, in bronchopneumonia, 466; in acute cardiac dilatation, 498; in diphtheria, 307; in lobar pneumonia, 450; in scarlet fever, 329; in typhoid fever, 277.
Branchial cysts, 158.
Breast feeding, 112; importance of, to premature infant, 67; mixture of other food with, 114; normal method of, 112; weaning from, 116; wet nurse for, 117.
Breast milk. *See Milk, human.*
Breasts, swelling and tenderness of, as complication of mumps, 359.
Breathing exercises, 53.

Breck, feeding tube devised by, 68.
Brewer's method of drainage in empyema, 480.
Bright's disease, as complication of bronchopneumonia, 460; as complication of measles, 336. *See also Acute nephritis.*
Broadbent, on irregular fever resisting quinin suggesting acute endocarditis, 490; on exaggeration of diastolic shock in chronic pericarditis, 514.
Bromid of potash, use of, in asthma, 712; in acute catarrhal bronchitis, 441; in bronchopneumonia, 467; in chorea, 701; in diphtheria, 306; in enuresis, 603; in epilepsy, 695; in influenza, 314; in acute laryngitis, 432; in lobar pneumonia, 453; in measles, 339; in acute nephritis, 581; in pavor nocturnus, 675; in pseudo-masturbation, 609; in typhoid fever, 278; in variola, 348; in urticaria, 727; in whooping cough, 294.
Bromid of soda, use of, in influenza, 314; in measles, 339; in pavor nocturnus, 675; in acute pericarditis, 513; in poliomyelitis, 651.
Bromids, use of, in cough of tuberculosis, 400; in acute endocarditis, 493; in functional heart disorders, 509; in headache, 710; in hysteria, 708; in insomnia, 677; in meningococcic meningitis, 638; in acute nephritis, 581; in multiple neuritis, 664; in nystagmus and head-nodding, 691; in purulent meningitis, 641; in tetany, 689; in thyroid intoxication, 558.
Bronchi, foreign bodies in, 435.
Bronchitis, acute catarrhal, 438; age as predisposing factor, 439; complications of, 440; definition of, 438; diagnosis of, 440; duration of, 440; etiology of, 438; fever in, 439; microorganisms present in, 438; mortality from, in young children, 2; pathology of, 439; physical signs of, 440; prognosis of, 440; prophylaxis in, 441; sources of infection in, 438; symptomatology of 439; treatment of, 441.
Bronchitis, afebrile asthmatic, 440.
Bronchitis, chronic, 442.
Bronchitis, membranous, 442.
Bronchopneumonia, 454; abortive type of, 461; as complication of acute catarrhal bronchitis, 438, 440; as complication of congenital atelectasis, 74; as complication of diphtheria, 300, 307; as complication of influenza, 312; as complication of measles, 336, 338; as complication of whooping cough, 455; complications of, 462; counterirritation in treatment of, 468; deglutition type of, 462; diagnosis of, 463; dietetic treatment of, 465; etiology of, 454; gastroenteritis followed by, 462; hygienic treatment of, 465; medical treatment of, 466; microorganisms present in, 454, 455; mortality from, 464; in new-born, 461; pathology of, 455; physical signs in, 460; poultices in treatment of, 468; predisposing causes of, 454; prognosis of, 464; prophylaxis of, 464; pulse rate in, 457; sources of infection in, 455; sputum findings in, 460; symptomatology of, 456; temperature curve in, 457, 458; treatment of, 464; treatment of during convalescence, 469; tuberculous form of, 386, 462; types of, 461; urine findings in, 459.
Broths, use of, in diet of infancy, 129, 142; in diet of typhoid fever, 277.
Brudzinski, on proteus vulgaris in causation of enteric infection, 178.
Bryant's method of drainage in empyema, 480.
Budin, on the influence of demand on supply in breast milk, 113.
Buhl's disease. *See Acute fatty degeneration of new-born.*
Burk, measurements of growth in children by, 17.
Buttermilk, use of, in artificial feeding of infants, 122; in chronic gastritis, 172; in enteric infection, 194; in infantile atrophy, 201; in typhoid fever, 277.

## C

Caffein citrate, for relief of headache, 710.
Caffein sodium benzoate, therapeutic use of, in acute cardiac dilatation, 498; in diphtheria, 307; in lobar pneumonia, 453; in poliomyelitis, 651; in scarlet fever, 330.
Caffein sodium salicylate, hypodermic use of, in splanchnic paralysis of diphtheria, 307.
Calcium, presence of, in milk, 104; uses of, to body, 105.
Calcium salts, therapeutic use of, in furunculosis, 729; in hemophilia, 540; in purpura, 536; in sporadic cretinism, 556; in tetany, 689.

CALOMEL, therapeutic use of, in acidouuria, 567; in acute articular rheumatism, 405; in combination with santonin for relief of ascaris lumbricoides, 216; in chorea, 700; in chronic constipation, 207; in enteric infection, 193; in erythema multiforme, 730; in acute gastric indigestion, 160; in acute gastritis, 162; in gastroduodenitis, 165; in headache, 710; in influenza, 313; in intestinal indigestion, 187; in lobar pneumonia, 452; in malaria, 266; in multiple neuritis, 663; in combination with santonin for relief of oxyuris vermicularis, 218; in poliomyelitis, 651; in purpura, 536; in recurrent vomiting, 258; in scarlet fever, 329; in syphilis externally, 373; in syphilis internally, 370, 371; in tetany, 689; in tonsillitis, 422; in typhoid fever, 277.

CALOMEL, use of, by sublimation, in laryngeal diphtheria, 304.

CALORIC STANDARD IN ARTIFICIAL FEEDING OF INFANTS, 130.

CAMERER AND SÖLDNER, on composition of colostrum, 108.

CAMPHOR, therapeutic use of, in acute cardiac dilatation, 498.

CAMPHORATED OIL, therapeutic use of, as counterirritant in bronchopneumonia, 468; to bring out eruption in measles, 340.

CANCRUM ORIS. *See Stomatitis gangrenosa.*

CANE SUGAR, use of, in artificial feeding of infants, 125.

CANNABIS INDICA, therapeutic use of, in epilepsy, 696.

CANTHARIDES, as cause of hematuria, 563; as cause of toxic nephritis, 573, 575.

CARBOHYDRATE DILUENTS, 138.

CARBOHYDRATES, 103, 124; acid intoxication due to deficient absorption of, 103; addition of, to skimmed milk, 124; use of, in artificial food, 135; indigestion due to deficiency of, 104; indigestion due to excess of, 104; food value of, 103, 138; food value of, in acetonuria, 565; food value of, in diabetes, 250, 251; food value of, in intestinal disorders, 178; proportion of, in buttermilk, 122, 123; proportion of, in Finkelstein's albumin milk, 123; proportion of, in milk, 103; proportion of, in modified milk mixture, 124; skimmed milk in relation to, 124; symptoms of over-feeding with, 184, 186.

CARBOLIC ACID OINTMENT, use of, in erythema multiforme, 730; in measles, 339.

CARBONATE OF POTASH. *See Potassium carbonate.*

CARDIAC DILATATION, ACUTE, 496; diagnosis of, 496; diagnosis between pericarditis and, 511; in diphtheria, 296, 297; etiology of, 496; in influenza, 312, 314; prognosis of, 496; prophylaxis of, 497; treatment of, 498; in whooping cough, 290.

CARDIAC DISEASES. *See under Heart.*

CARDIAC COMPENSATION, acute cardiac dilatation due to failure of, 496; in mitral regurgitation, 499; in mitral stenosis, 500; failure of, in chronic valvular heart disease, 502; treatment of, 505; tricuspid regurgitation due to failure of, 502.

CARDIAC MURMURS. *See Murmurs.*

CARDIAC PARALYSIS, in diphtheria, 299; in multiple neuritis, 663.

CARIES OF SPINE. *See Pott's disease.*

"CARRIERS:" of diphtheria, 295; of tuberculosis, 373; of typhoid fever, 267, 268.

CASCARA, therapeutic use of, in constipation, 207; in measles, 340; in acute nephritis, 579; in scarlet fever, 329.

CASEIN, action of alkalies on, 139; composition of, 102; excess of, injurious, 135; inability to digest, 186; proportion of, in buttermilk, 123; proportion of, in milk, 102.

CASTOR-OIL, therapeutic use of, in acute catarrhal bronchitis, 442; in bronchopneumonia, 466; in chorea, 700; in eclampsia, 682; in enteric infection, 196; in erythema multiforme, 730; in fever, 266; in headache, 710; in Henoch's purpura, 537; in acute intestinal indigestion, 187, 188; in lobar pneumonia, 452; in measles, 340; in multiple neuritis, 663; in poliomyelitis, 651; in pseudo-leukemia, 526; in stomatitis catarrhalis, 147; in stomatitis aphthosa, 148; in stomatitis mycosa, 150; in tonsillitis, 422; in typhoid fever, 277; in urticaria, 727.

CASTOR-OIL, use of, contraindicated, in constipation of infants, 204, 206; in acute gastric indigestion, 160; in acute gastritis, 162.

CATARRH, ACUTE NASAL. *See Rhinitis, acute.*

CATARRHAL JAUNDICE. *See Gastroduodenitis.*

CATHARTICS, therapeutic use of, in chorea, 700; in eclampsia, 683; in erythema multiforme, 730; in headache, 709; in lobar pneumonia, 452; in measles, 340; in acute nephritis, 578; in toxic nephritis, 574; in pavor nocturnus, 675; in purpura, 536; in urticaria, 727.

CATHETER, form of, for use in gonorrheal vulvovaginitis, 594; importance of sterilizing, in myelitis, 656.

CATHETERIZATION, indications for, in infant, 559; necessity for, in myelitis, 656.

CELLULITIS, as complication of vaccinia, 353, 354.

CEPHALHEMATOMA, IN NEW-BORN, 97.

CEREAL DECOCTIONS, in artificial feeding of infants, 126, 135; as medium for quinin, 285; in diet of typhoid fever, 277, 278.

CEREBRAL CROUP. *See Laryngismus stridulus.*

CEREBRAL HEMORRHAGE, as cause of epilepsy, 692; as complication of Henoch's purpura, 535.

CEREBROSPINAL FLUID, diagnostic value of, 37; in diagnosis between brain abscess and meningitis, 618; in meningococcus meningitis, 634; in poliomyelitis, 649; in purulent meningitis, 639; in tuberculous meningitis, 629.

CEREBROSPINAL MENINGITIS. *See Meningitis, meningococcus.*

CESTODES, INTESTINAL, 211; ascaris lumbricoides, 215; bothriocephalus latus, 212; hymenolepis nana, 212; oxyuris vermicularis, 217; tenia elliptica, 213; tenia saginata, 211; tenia solium, 211.

CHAPIN, on albuminuria in pulmonary disease, 572; on education of infant stomach, 122; on gastrointestinal disorders, as cause of toxic nephritis, 572; measurements of growth in children by, 17.

CHAPIN'S INFANT URINAL, 559.

CHART, showing temperature curve, in bronchopneumonia (mild), 459; in bronchopneumonia (severe), 460; in congenital malaria, 282; in empyema, 474; in empyema, following pleurisy, 473; in gastroenteric infection (mild), 189; in gastroenteric infection (severe), 190; in laryngeal diphtheria treated with antitoxin and intubation, 298; in lobar pneumonia in child two years old, 445; in lobar pneumonia in child four years old, 446; in lobar pneumonia in child ten years old, 447; in measles (uncomplicated), 336; in measles, complicated with bronchopneumonia, 337, 338; in meningococcic meningitis in child six years old, 634; in pharyngeal diphtheria treated with antitoxin, 297; in anterior poliomyelitis of bulbar type, 446; in purulent meningitis in child ten years old, 640; in scarlet fever in child six years old, 320; in scarlet fever in child twelve years old, 319; in tuberculous meningitis in child twenty months old, 629; in typhoid fever in child two and a half years old, 270; in typhoid fever in child six years old, 271; in typhoid fever in child ten years old, 272; in typhoid fever relapse in child three years old, 274.

CHART, showing, blood changes in dermatitis exfoliativa in new-born, 79; blood changes in purpura hemorrhagica, 534; mortality in diphtheria when treated with antitoxin, 303; mortality by age in scarlet fever, 326; ratio of increase in weight in dull, mediocre, and precocious children, 16; weight in artificially fed infants, 136; weight in breast-fed infants, 114.

CHEADLE, on acute articular rheumatism, 402, 489; on relation between chorea and endocarditis, 697.

CHEST, palpation of, 30.

CHEYNE-STOKES RESPIRATION, in meningococcic meningitis, 633; in tuberculous meningitis, 628.

CHITTENDEN, analysis by, of Nestlé's food, 127.

CHICKEN-POX. *See Varicella.*

CHILD CROWING. *See Laryngismus stridulus.*

CHILDHOOD, excessive nerve activity in, 6; heat-dissipating mechanism in, 26; heat-regulating mechanism in, 25; inhibitory function undeveloped in, 24; irritability of nervous system in, 24; mortality in, 1, 2; muscular development in, 20; nervous system in, 23; peculiarities of blood in, 518; rest essential to health in, 11; sleep in, 13; temperature in, easily affected, 26; weight important in, 13, 16.

CHLORAL, therapeutic use of, in asthma, 712; in chorea, 701; in cough of measles, 339; in cough of tuberculosis, 400; in eclampsia, 683; in epilepsy,

696; in meningococcic meningitis, 638; in acute nephritis, 581; in purulent meningitis, 641; in tetanus neonatorum, 92; in tetany, 689; in variola, 348; in whooping cough, 294.
CHLORANEMIA, 545, 547; treatment of, 550.
CHLORATE OF POTASH. *See Potassium chlorate.*
CHLORID OF SODA. *See Sodium chlorid.*
CHLOROFORM, inhalation of, in asthma, 712; in chorea, 701; in diagnosis between laryngeal diphtheria and spasmodic catarrhal croup, 302; in eclampsia, 682; in epilepsy, 695; in laryngismus stridulus, 686; in acute laryngitis, 433; in acute nephritis, 581; danger from, in status lymphaticus, 547.
CHLOROSIS, 526; blood changes in, 527; color index of, 526; definition of, 526; diagnosis of, 527; diet in, 527; drugs in treatment of, 527; etiology of, 527; predisposing causes of, 527; prognosis of, 527; symptomatology of, 527; treatment of, 527.
CHOLERA INFANTUM, 192; heat as cause of, 262; hypodermic medication in, 196.
CHOREA, 696; in acute articular rheumatism, 404; cardiac murmurs in, 700; definition of, 696; diet in, 700; drugs in treatment of, 700; duration of, 698; endocarditis associated with, 489; etiology of, 697; exciting causes of, 697; general treatment of, 700; hygienic measures in, 701; medical treatment of, 700; predisposing causes of, 697; prognosis of, 698; symptomatology of, 698; treatment of, 700; urine findings in, 700.
CHOREA MINOR. *See Chorea.*
CHRYSAROBIN, therapeutic use of, in tinea tonsurans, 734.
CHVOSTEK, on causation of tetany, 687.
CHVOSTEK'S SYMPTOM, 688, 689.
CIRCUMCISION, for paraphimosis, 597; for phimosis, 596, 601.
CITRATE OF POTASH. *See Potassium citrate.*
CLARK, ANDREW, on constipation in relation to chorea, 527.
CLEFT PALATE, 156.
CLIMATE, influence of, in causation, of acute articular rheumatism, 402; of influenza, 308; of poliomyelitis, 643.
CLIMATE, influence of, in treatment, of asthma, 713; of chorea, 701; of endocarditis in rheumatic subjects, 493; of hemoglobinuria, 565; of heart disease, due to rheumatism, 503; of chronic influenza, 314; of chronic nephritis, 583; of recurrent vomiting, 257; of tuberculosis, 396, 401.
CLOUSTON, on difference between brain in childhood and maturity, 23.
CLUBBED FINGERS, in congenital heart disease, 486.
COAL-OIL, therapeutic use of, in pediculosis capillitii, 737.
COAL-TARS, use of, contraindicated, in chronic headache, 710; in lobar pneumonia, 451; in thyroid intoxication, 558; in typhoid fever, 278.
CODEIN, therapeutic use of, in cough of influenza, 314; in cough of measles, 339; in lobar pneumonia, 453; in pleurisy, 478; in paroxysms of whooping cough, 294; in tumor of kidney, 589; in variola, 348.
COD-LIVER OIL, therapeutic use of, in acute articular rheumatism, 406; in asthma, 713; in chronic bronchitis, 442; in chorea, 701; in constipation, 207; in convalescence from bronchopneumonia, 469; in convalescence from influenza, 315; in convalescence from acute laryngitis, 433; in convalescence from lobar pneumonia, 453; in enuresis, 601; in facial paralysis, 666; in laryngismus stridulus, 686; in multiple neuritis, 664; in nystagmus and head-nodding, 691; in pavor nocturnus, 675; in pseudo-masturbation, 609; in rickets, 244; in scurvy, 250; in simple secondary anemia, 524; in status lymphaticus, 550; in tetany, 689; in tuberculosis, 399.
COIT, on examination of milk, 118.
COLLARGOLUM, therapeutic use of, in scarlet fever, 330; in ulcerative endocarditis, 494; method for, 331.
COLLARGUM, suppositories of, 52.
COLLES' LAW, 361.
COLLODION, FLEXIBLE, use of, in adenitis, 544; in adenitis in scarlet fever, 331; in tinea tonsurans, 735.
COLD, EXPOSURE TO, a cause of hemoglobinuria, 565; of nephritis, 575.
COLD BATHS. *See Baths.*
COLD COMPRESS. *See Compress.*
COLD PACKS. *See Packs.*
COLI VACCINE, therapeutic use of, 59.
COLON BACILLUS, as cause, of cystopyelitis, 584; of enuresis, 601; of fever resembling typhoid, 267; of simple vulvovaginitis, 595.

Colon, congenital dilatation of, 208; etiology of, 208; prognosis of, 209; symptomatology of, 209; treatment of, 209.
Color index of blood, in chlorosis, 526; in pernicious anemia, 528; in simple anemia, 523; significance of, 516.
Colostrum, 107.
Coma, in acetonuria, 566; in meningococcic meningitis, 633; in acute nephritis, 576; in purulent meningitis, 639; in tuberculous meningitis, 628.
Comby, on antitoxin in post-diphtheritic paralysis, 307; on congenital mumps, 358; on relapses in typhoid fever, 274; on respiratory manifestations in lithemia, 711.
Compensation, cardiac. *See Cardiac.*
Compound licorice powder, 579.
Compress, cold, 46; hot, 46.
Condensed milk. *See Milk.*
Condylomata, in hereditary syphilis, 364.
Congenital hypertrophy of the pylorus, 166; diagnosis of, 168; etiology of, 166; pathology of, 166; prognosis of, 169; symptomatology of, 167; treatment of, 169.
Congenital ichthyosis, 730; treatment of, 731.
Conjunctival tuberculin test. *See Tuberculin.*
Conjunctivitis, in gonorrheal vaginitis, 593; in influenza, 312; in measles, 338; in acute rhinitis, 413.
Connors, J. F., on food percentages, 139.
Consanguinity, relation to, in parents to congenital idiocy, 623.
Constipation, in appendicitis, 224; in congenital dilatation of the colon, 208; in congenital hypertrophy of pylorus, 167; in enteric infection, 191; associated with enuresis, 603; due to Finkelstein's albumin milk, 124; in chronic gastritis, 171; in gastroduodenitis, 165; in icterus neonatorum, 94; in intestinal indigestion of infants, 198; in intestinal indigestion of older children, 202; in malaria, 286; in peritonitis, 231; in recurrent vomiting, 253; rectal suppositories in treatment of, 206; in sporadic cretinism, 554; due to sterilized milk, 120; in typhoid fever, 272; treatment of, in typhoid fever, 278.
Constipation, chronic, 204; diagnosis of, 205; dietetic causes of, 204; dietetic treatment of, in first year, 206; dietetic treatment of, in second year, 207; etiology of, 204; medical treatment of, 207; symptomatology of, 205; treatment of, in infancy, 206; treatment of, in older children, 207.
Convulsions, in cystopyelitis, 585; in epilepsy, 694; in hemiplegia, 612; in malaria, 284; in uremia, due to acute nephritis, 576.
Coryza, acute, *see Acute rhinitis;* danger from, in infancy, 6; in influenza, 310; treatment of, in influenza, 313; recurrent, 258; syphilitic, 362, 363.
Cough, character of, in bronchopneumonia, 457; in acute catarrhal bronchitis, 439; in influenza, 311; in laryngeal diphtheria, 298; in acute laryngitis, 430; in lobar pneumonia, 447; in measles, 334; in pleurisy, 473; in retropharyngeal abscess, 428; in acute rhinitis, 413; in enlarged thymus, 546; in tuberculosis, 400; in whooping cough, 289.
Cough syrups, contraindicated, in acute bronchitis, 441; in bronchopneumonia, 467.
Councilman, on "cytorrhyctes variolæ," 345; on vesicular fluid in chicken-pox, 356.
Counterirritation, use of, in bronchopneumonia, 468; in facial paralysis, 666; in lobar pneumonia, 451; objections to, in acute pericarditis, 512; in chronic pericarditis, 513; in pleurisy, 478.
Coutts, on night-terror, 673, 674.
Cowan, on mortality from small-pox before vaccination, 350.
Cow's milk. *See Milk.*
Cow-pox. *See Vaccinia.*
Crandall, on congenital malaria, 281; on infantile scurvy, 246; on vaccination, 349.
Cream, as source of fat in artificial feeding, 135; use of, in constipation, 206; percentage of, in modified milk, 137.
Cream mixtures, furnished by Walker-Gordon Laboratories, 141; objections to use of, in constipation, 206; injury due to excess of, 186.
Creosote, therapeutic use of, in chronic bronchitis, 442; in convalescence from bronchopneumonia, 469; in lobar pneumonia, 452; in tuberculosis, 398; by inhalation in measles with septic complications, 340.
Cresolin, inhalations of, in rhinitis, 414; in whooping cough, 293.
Cretinism, endemic, 553.

CRETINISM, SPORADIC, 552; definition of, 553; diagnosis of, from Mongolian idiocy, 554; diagnosis of, from thyroid insufficiency, 555; etiology of, 553; prognosis of, 555; treatment of, 555; varieties of, 552.
CROCKER, on treatment of tinea tonsurans, 735.
CROUP, cerebral, *see Laryngismus stridulus;* false, *see Acute laryngitis;* membranous, *see Laryngeal diphtheria.*
CROUPOUS PNEUMONIA. *See Lobar pneumonia.*
CROUP TENT, use of, in acute laryngitis, 433.
CURVATURE OF SPINE, in school children, 19; in sporadic cretinism, 554.
CYANOSIS, in bronchopneumonia, 458; following cerebral injury at birth, 612; in congenital heart disease, 485; in diagnosis between broncho- and lobar pneumonia, 464; in edema of the larynx, 434; in acute laryngitis, 431; in multiple neuritis, 663; in myocarditis, 495; oxygen for relief of, 488; in paroxysmal hemoglobinuria, 565; in pericarditis, 510; in premature infants, 65; in stenosis of pulmonary artery, 487; in thyroid enlargement, 546; in valvular heart disease with failing compensation, 502.
CYCLIC ALBUMINURIA. *See Orthostatic albuminuria.*
CYSTITIS, as complication in myelitis, 656.
CYSTOPYELITIS, 583; age as predisposing factor in, 584; definition of, 583; diagnosis of, 585; drugs in treatment of, 587; etiology of, 584; microorganisms present in, 584, 586; mortality from, 586; operative treatment for, 587; prognosis of, 586; prophylaxis of, 586; recurrent vomiting in, 585; sex as predisposing cause of, 584; sources of infection in, 584; symptomatology of, 585; temperature range in, 585; termination of, 586; treatment of, 586; tumor in, 585; urine findings in, 586; X-ray picture in, 586, 587.
"CYTORRHYCTES VARIOLÆ," 345.
CZERNY, on hydrocephalus and diseases of adrenals, 619; on over-feeding a cause of intestinal indigestion, 174, 175; on over-feeding injurious to metabolism, 175; on symptom group in acute intestinal indigestion, 184.

D

DACTYLITIS, 393.
DEAFNESS, due to adenoid growths, 425; following use of Flexner serum, 638; following meningococcic meningitis, 635; due to mumps, 359; due to syphilis, 367.
DEATH, causes of, in infancy and childhood, 1.
DEBILITY, general, nursing infant contraindicated by, 112.
DEFECTIVE INTERVENTRICULAR SEPTUM, 487.
DEGLUTITION PNEUMONIA, 462.
DENTITION, delayed, as a pathological factor, 145; first, as a cause of intestinal disturbances, 176.
DERMATITIS, in new-born, 79; treatment of, 80.
DERMATITIS EXFOLIATIVA, 79.
DESQUAMATION, in measles, 336; in scarlet fever, 322, 327; in variola, 347.
DEW'S METHOD OF ARTIFICIAL RESPIRATION, 73.
DEXTRIN, combination of, with maltose in artificial food, 125.
DEXTRINIZED GRUELS, use of, in artificial feeding, 126.
DIABETES MELLITUS, 250; toxic nephritis associated with, 572.
DIACETIC ACID, presence of, in urine in acetonuria, 565; presence of, in urine in measles, 336; test for, with ferrous chlorid, 566.
DIARRHEA, in cholera infantum, 192; in enteric infection, 191; in acute intestinal indigestion, 183, 186; in chronic intestinal indigestion, 198; in miliary tuberculosis, 387; in peritonitis, 231; in tuberculosis of lymph-nodes, 383; treatment of, in tuberculosis, 400; in typhoid fever, 272, 278.
DIAZO REACTION OF EHRLICH, 273, 275, 336; absence of, in rubella, 343.
DICKIE, operation by, for obstruction of esophagus, 158.
DIET, as a means of controlling putrefactive processes in intestinal canal of infants, 177.
DIET, ERRORS IN, a cause, of constipation, 204, 206; of edema, 578; of acute gastric indigestion, 159; of recurrent vomiting, 251; of rickets, 237; of scurvy, 246.
DIET, therapeutic value of, in acute articular rheumatism, 405, 406; in appendicitis, 227; in bronchopneumonia, 465; in chlorosis, 527; in chorea, 700; in congenital hypertrophy of pylorus, 170; in constipation, 206, 207; in diabetes,

250; in dilatation of the stomach, 164; in diphtheria, 307; in eclampsia, 684; in eczema, 722; in edema of glottis, 435; in acute endocarditis, 493; in enteric infection, 193; in erythema multiforme, 730; in first year of life, 142; in fourth to sixth year of life, 143; in functional heart disease, 508; in acute gastric indigestion, 160; in acute gastritis, 162; in chronic gastritis, 172; in gastroduodenitis, 166; in infantile cerebral palsies, 615; in acute infectious disease, as prophylaxis against nephritis, 578; in acute intestinal indigestion, 188; in chronic intestinal indigestion of infants, 200; in chronic intestinal indigestion of older children, 203; in lobar pneumonia, 450; in malaria, 286; in measles, 339; in meningococcic meningitis, 638; in multiple neuritis, 663; in mumps, 360; in acute nephritis, 580; in chronic nephritis, 583; in nystagmus and head-nodding, 691; in obscure fevers, 266; in orthostatic albuminuria, 570; in pavor nocturnus, 675; in acute pericarditis, 512; in peritonitis, 231; in pica, 705; in purpura, 535; in recurrent vomiting, 257; in rickets, 243; in scarlet fever, 329; in scurvy, 249; in second year of life, 143; in simple secondary anemia, 527; in status lymphaticus, 550; in stomatitis aphthosa, 148; in stomatitis catarrhalis, 147; in stomatitis mycosa, 150; in syphilis, 369; in tetany, 689; in third year of life, 143; in tonsillitis, 423; in tuberculosis, 397; in tuberculous peritonitis, 401; in typhoid fever, 276; in urticaria, 728; in valvular disease of the heart, 504; in varicella, 357; in variola, 348; in whooping-cough, 292.

Digestive ferments, 125; in milk, 106.

Digital examination, 30.

Dilatation of stomach, 163; diagnosis of, 163; etiology of, 163; prognosis of, 164; symptomatology, 163; treatment, 164.

Diluents, alkaline, 139; carbohydrates, 138.

Diphtheria, 295; antitoxin in treatment of, *see Antitoxin;* bacillus of, 295; bacillus of, in cystopyelitis, 584; blood picture in, 297; "carriers" of, 295; complications of, 299; course of, 300; diagnosis of, 301; diagnosis between, and spasmodic catarrhal croup, 301; diet in, 307; drugs in, 307; duration of, 300; etiology of, 295; of the eye, 299; immunity from, 295; incubation period of, 296; intubation in, 304, 305; of larynx, 297; local treatment of laryngeal form, 304; measles complicated by, 338; mortality from, 2, 300; multiple neuritis due to, 299; myocarditis as complication of, 299; paralysis following, 299, 307; pathology, 296; predisposing causes of, 295; prognosis of, 300; prophylaxis in, 302; quarantine in, 302; septicemia as complication of, 299, 307; symptomatology of, 296; temperature curve in, 297; toxemia in, 299; tracheotomy in, 304; transmission of, 295; of vulva, 299.

Diplegia, 614; treatment of, 615.

Diuretics, use of, in pleurisy with effusion, 478; contraindicated in acute nephritis, 581.

Discharges, gastrointestinal in newborn, 3; from intestinal tract in intussusception, 220.

Diuretin, use of, in pleurisy with effusion, 478.

Douche, nasal, 48.

Dropsy. *See Ascites* and *Edema.*

Drugs, effect of, upon milk of nursing mother, 112; hematuria caused by, 563; hemoglobinuria caused by, 564; purpura caused by, 532; toxic nephritis caused by, 573; suited for use by inunction, 42; suited for use by mouth, 38.

Drugs, employed in treatment of, acute articular rheumatism, 405; of asthma during attack, 712; of asthma during interval, 713; of bronchopneumonia, 466; of acute catarrhal bronchitis, 441; of acute cardiac dilatation, 498; of chlorosis, 528; of chorea, 700; of congenital dilatation of colon, 210; of congenital ichthyosis, 731; of constipation, 207; of dilatation of stomach, 164; of diphtheria, 307; of eclampsia, 683; of eczema, 723; of acute endocarditis, 494; of enteric infection, 195; of enuresis, 602; of epilepsy, 696; of functional heart disease, 508; of furunculosis, 729; of acute gastric indigestion, 160; of acute gastritis, 162; of chronic gastritis, 173; of gastroduodenitis, 165; of headache, 710; of hemophilia, 539; of influenza, 315; of intestinal indigestion in infants, 201; of intestinal indigestion in older chil-

dren, 203; of acute laryngitis, 432; of lobar pneumonia, 452; of measles, 339; of multiple neuritis, 664; of mumps, 360; of acute nephritis, 579; of pericarditis, 513; of pernicious anemia, 529; of pleurisy, 478; of poliomyelitis, 651; of pseudo-masturbation, 609; of rachitis, 244; of recurrent vomiting, 256; of acute rhinitis, 414; of simple anemia, 524; of sporadic cretinism, 556; of status lymphaticus, 550; of stomatitis aphthosa, 148; of stomatitis catarrhalis, 147; of stomatitis ulcerosa, 151; of syphilis, 370; of tinea tonsurans, 734; of tonsillitis, 422; of tuberculosis, 398; of urticaria, 727; of valvular heart disease, 504; of variola, 348; of whooping cough, 294.

DRY CUPPING, in treatment of acute nephritis, 580.

DUCHENNE-ERB'S PALSY. *See* Upper-arm paralysis.

DUCTLESS GLANDS, enlargement of, in status lymphaticus, 545.

DUNN, on favorable influence of lumbar puncture in tuberculous meningitis, 629; on value of cerebrospinal fluid in diagnosis of tuberculous meningitis, 629.

DWARFISHNESS, association of, with endemic cretinism, 553; with sporadic cretinism, 553, 554; with concealed tuberculosis, 383.

DYSPNEA, presence of, in acetonuria, 566; in asthma, 711; in bronchopneumonia, 457, 458; in acute catarrhal bronchitis, 440; in congenital dilatation of colon, 209; in congenital heart disease, 485; in congenital laryngeal stridor, 437; in edema of larynx, 434; in acute endocarditis, 490; in Hodgkin's disease, 541; in laryngeal diphtheria, 299; in acute laryngitis, 430, 431; in multiple neuritis, 663; in myocarditis, 495; in pericarditis, 510; in periesophageal abscess, 158; in pleurisy, 473; in retropharyngeal abscess, 428; in status lymphaticus, 545, 546; in tuberculous bronchopneumonia, 462; in tuberculosis of bronchial lymph-nodes, 382; in tuberculosis of mesenteric lymph-nodes, 383.

DYSTROPHY, PROGRESSIVE MUSCULAR, 666.

E

EARACHE, due to adenoid growths, 425; due to otitis media, 715, 716; fever associated with, 262, 263, 264; significance of, in bronchopneumonia, 463.

EBERTH, description of typhoid bacillus by, 267.

ECLAMPSIA, 678; definition of, 678; diet in, 684; due to epilepsy, 680; etiology of, 678; exciting causes of, 680; hysterical form of, 707; idiocy due to, 623; predisposing causes of, 678; prognosis of, 681; reflex causes of, 680; symptomatology of, 680; treatment of, 682.

ECZEMA, 719; crustosum, 721; definition of, 719; diagnosis of, 721; diet in, 722; etiology of, 719; external causes of, 720; general treatment of, 721; internal causes of, 719; local applications in, 723; papular form of, 720; prognosis of, 721; pustular form of, 721; rubrum, 721; squamosum, 721; symptomatology of, 720; treatment of, 721.

EDEBOHLS' OPERATION, for splitting capsule of kidney, 583.

EDEMA, of glottis, *see Edema of larynx;* idiopathic form of, 578; of larynx, 434; in acute nephritis, 576, 578; in tetany, 688.

EDEMA, LOCALIZED, in Henoch's purpura, 534; in purpura rheumatica, 535.

EDSAL, on bicarbonate of soda in recurrent vomiting, 256.

EGG ALBUMIN, use of, in artificial feeding of infants, 137.

EGGS, SOFT-BOILED, in diet of infants, 142; in diet of typhoid fever, 277.

EHRLICH'S DIAZO-REACTION. *See Diazo-reaction.*

ELA, on manipulations in artificial respiration, 73.

ELECTRIC HEATERS, use of, in acute nephritis, 580.

ELECTRICITY, therapeutic use of, in congenital dilatation of colon, 210; in facial paralysis, 666; in hysteria, 708; in multiple neuritis, 664; in poliomyelitis, 652; in post-diphtheritic paralysis, 309; in progressive muscular atrophy, 669.

EMETIC, value of, in acute laryngitis, 432.

EMPHYSEMA, association of, with asthma, 712; with whooping cough, 289.

EMPYEMA, bilateral, 481; Brewer's method of drainage in, 480; Bryant's method of drainage in, 480; bronchopneumonia complicated by, 462;

chronic form of, 481; pathology of, 471; pneumococcus in causation of, 470; respiratory capacity diminished in, 471; surgical treatment of, 479; temperature curve in, 472; treatment of, during convalescence, 482.
ENCEPHALITIS, a cause of idiocy, 623.
ENCEPHALOCELE, 621.
ENDOCARDITIS, ACUTE, 489; in acute articular rheumatism, 404; in chorea, 697; definition of, 489; diagnosis of, 492; diet in, 493; drugs in treatment of, 493; etiology of, 489; in erythema multiforme, 730; in fetal life a cause of congenital heart disease, 485; heredity in causation of, 489; in Henoch's purpura, 534; in lobar pneumonia, 448; microorganisms present in, 490; pathology of, 490; physical signs of, 491; prognosis of, 492; prophylaxis of, 492; recurrent attacks of, associated with rheumatism, 503; septic form of, 491; sources of infection in, 490; symptomatology of, 490; temperature curve in, 490; tonsillitis associated with, 422; treatment of, 493; ulcerative form of, 494.
ENEMATA, HIGH, for relief, of eclampsia, 683; of intestinal intussusception, 222; of oxyuris vermicularis, 218; of recurrent vomiting, 256.
ENEMATA, RECTAL, in congenital dilatation of colon, 210; in chronic constipation, 207; in acute intestinal indigestion, 187; in chronic intestinal indigestion, 203; in measles, 340; in peritonitis, 232; therapeutic value of, 51; in tuberculous peritonitis, 401; in typhoid fever, 278.
ENTERIC INFECTION, 189; cholera infantum a form of, 192; definition of, 189; diarrhea in, 191; diet in, 193; drugs in, 195; hygienic treatment of, 197; medicinal treatment of, 195; nervous symptoms in, 192; pathology of, 189; prognosis in, 193; stimulating treatment of, 196; symptomatology of, 190; temperature curve in, 190, 192; treatment of, 193.
ENURESIS, 598; belladonna in treatment of, 602; diet in treatment of, 601; drugs in treatment of, 602; etiology of, 599; exciting causes of, 599; factors in, 598, 599; general treatment of, 601; habit in causation of, 599; heredity in causation of, 599; malnutrition in causation of, 599; medicinal treatment of, 602; physiology of, 598; predisposing causes of, 599; prognosis of, 601; reflex irritation as cause of, 599, 601; symptomatology of, 601; treatment of, 602; urine findings in, 601.
ENVIRONMENT, as cause, of hysteria, 706; of masturbation, 606; of pseudomasturbation, 606.
ENTEROCOLITIS, association of, with scurvy, 248; as complication of influenza, 310; as complication of measles, 338; as complication of whooping-cough, 290; diagnosis between, and intestinal intussusception, 221; *See also Enteric infection.*
ENTERITIS, as complication of diphtheria, 300.
EPILEPSY, 691; aura in, 693; convulsions in, 694; definition of, 691; diagnosis of, 694; diet in, 695; diplegia a cause of, 614; drugs in treatment of, 696; eclampsia due to, 680; etiology of, 691; hemiplegia associated with, 613; idiocy due to, 623; loss of consciousness in, 693; meningeal hemorrhage a cause of, 611; mental symptoms of, 694; number of attacks in, 693; prognosis in, 694; pseudomasturbation not related to, 607; suggestion in treatment of, 696; surgical treatment of, 696; symptomatology of, 692; treatment of, 695; types of, 692.
EPISPADIAS, 598.
EPISTAXIS, 415; in adenoid disease, 425; etiology of, 415; in leukemia, 530; in mumps, 359; predisposing causes in, 415; in purpura hemorrhagica, 533; treatment of, 416; in typhoid fever, 273.
EPSOM SALTS. *See Magnesium sulphate.*
ERB, on causes of tetany, 687.
ERB'S juvenile type of progressive muscular atrophy, 668.
ERB'S SYMPTOM, 688, 689.
ERGOT, purpura due to, 532.
ERUPTION, character of, in acute articular rheumatism, 404; in congenital ichthyosis, 730; from diphtheritic antitoxin, 304; in eczema, 720; in erythema infectiosum, 343; in erythema multiforme, 730; in Henoch's purpura, 534; in impetigo contagiosa, 731; in influenza, 310, 311; in measles, 335; in meningococcic meningitis, 634; in pemphigus neonatorum, 732; in purpura, 531; in purpura fulminans, 532; in purpura hemorrhagica, 533; in purpura rheumatica, 535; in purpura simplex, 532; in rubella, 342; in scabies,

735; in scarlet fever, 321; in symptomatic purpura, 532; in syphilis, 363, 364; in tinea tonsurans, 733; in urticaria, 726; in varicella, 355; in variola, 346.

ERUPTION, following use of diphtheritic antitoxin, 304; following influenza, 310, 311; method of bringing out in measles, 340; of pemphigoid type as complication of measles, 338.

ERYSIPELAS, in new-born, 80; as complication of vaccinia, 353.

ERYTHEMA, combined with eczema, 720; combined with Henoch's purpura, 534; combined with purpura rheumatica, 535.

ERYTHEMA INFECTIOSUM, 343.

ERYTHEMA MULTIFORME, 729; treatment of, 730.

ERYTHROBLASTS, 515.

ERYTHROCYTES. *See Red blood corpuscles.*

ESCHERICH, on "blue" bacillus in epidemic of enteric infection, 178; on colon bacillus as exciting cause of cystopyelitis, 584; on predisposing causes of tetany, 687; on streptococcus enteriditis in epidemic of acute enteric infection, 178; description of erythema infectiosum by, 343.

ESCHERICH AND MOSER, reduction of mortality in scarlet fever by, through use of Moser's serum, 330.

ESOPHAGITIS, 157.

ESOPHAGUS, congenital malformation of, 158.

ETHER, hypodermic use of, in diphtheria, 307.

ETHMOIDAL SINUSES, brain abscess due to septic infection of, 618; infection of, in diphtheria, 300; infection of, in influenza, 312.

EUCALYPTUS, use of, as spray in scarlet fever, 330.

EUCALYPTUS OIL, inhalation of, in measles with septic complications, 340; local use of, in influenza, 314.

EUQUININ, therapeutic use of, in influenza, 313; in lobar pneumonia, 452; in malaria, 285; in acute rhinitis, 415; in whooping-cough, 293.

EXAMINATION OF SICK CHILD, 28; by auscultation, 32; of blood, 37; of cerebrospinal fluid, 37; digital form of, 30; of extremities, 31; family history, 28; by inspection, 29; by palpation, 30; by percussion, 32; physical, 29; previous medical history, 28; of reflexes, 31; by Röntgen rays, 38; of spinal column, 31; of stools, 32; for tuberculosis, 34; of urine, 32.

EXERCISE, MUSCULAR, excess of, a cause of acute cardiac dilatation, 496; orthostatic albuminuria affected by, 570; passive forms of, useful in hemiplegia, 615; restriction of, in functional heart disease, 504; restriction of, in valvular heart disease, 502.

EXPECTORANTS, objections to use of, in bronchopneumonia, 467; in lobar pneumonia, 453; in measles, 340.

EXPECTORATION. *See Sputum.*

EXTREMITIES, examination of, in illness, 31.

EYE, DIPHTHERIA OF, 299; antitoxin in treatment of, 303; local treatment of, 304.

EYES, CARE OF, in measles, 340; in new-born, 3; in variola, 348.

EYE STRAIN, associated with epilepsy, 692; as cause of headache, 709.

## F

FACE AND HEAD, examination of, in illness, 29.

FACIAL EXPRESSION, in cretinism, 553; in idiocy, 623, 624; significance of, in illness, 29.

FACIAL PARALYSIS, 98.

FAMILY HISTORY, bearing of, on illness, 28.

FAT, food value of, 101, 103; indigestion due to excess of, 185; proportion of, in buttermilk, 122; proportion of, in condensed milk, 127; proportion of, in colostrum, 108; proportion of, in Finkelstein's albumin milk, 123; proportion of, in malted milk, 128; proportion of, in malt soups, 124; proportion of, in modified milk, 137; proportion of, in skimmed milk, 124; reduction of, advisable in diet during hot weather, 181; Walker-Gordon preparation furnishing, 137.

FATTY DEGENERATION OF NEW-BORN, 82.

FEDINSKI AND NICOLL, use of antistreptococcic serum by, 330.

FETUS, malaria in, 281; typhoid in, 269.

FEVER, exciting causes of, in infancy and childhood, 261; autoinfection of non-bacterial origin, 262; excessive muscular activity, 263; heat stroke, 262; inanition, in first week of life, 87, 263; intestinal toxemia, 261, 265; lobar pneumonia, 264; mechanical irritation, 262;

otitis media, 264; pyelocystitis, 264; reflex irritation, 262; septic infection, 265; sepsis in second week of life, 263; systemic intoxication of bacterial origin, 261; tuberculosis, 264; typhoid fever, 265.

Fevers, obscure, of infancy and childhood, 263; antipyretics in treatment of, 266; diet in, 266; inanition a cause of, 87, 263; intestinal toxemia a cause of, 261, 265; lobar pneumonia a cause of 264; otitis media, a cause of, 264; pyelocystitis a cause of, 264; septic infection a cause of, 265; typhoid fever a cause of, 265; tuberculosis a cause of, 264.

Fibrinous pneumonia. *See Lobar pneumonia.*

Filix mas. *See Aspidium.*

Finkelstein's albumin milk, 123; use of, in artificial feeding of infants, 123; in enteric infection, 194; in intestinal indigestion, 201.

Finkelstein and Meyer, on use of casein in intestinal indigestion, 102.

Fish tapeworm, 528, 529.

Fischer, Martin H., on formula for rectal injections in acute nephritis, 579; on mineral salts in treatment of paroxysmal hemoglobinuria, 565; on rectal injections in eclampsia, 684; on subcutaneous alkaline injections in uremia, 580; on value of water in infant feeding, 106.

Fissure of anus, 235.

Flexner, on Shiga bacillus, 178.

Flexner and Lewis, on inoculation of monkeys with poliomyelitis, 641.

Flexner serum, action of, 637; dose of, 636; results of, in meningococcus meningitis, 635, 637; results of, in purulent meningitis, 640; technique of administration, 636.

Flies, in transmission of acute anterior poliomyelitis, 642; in transmission of typhoid fever, 268.

Fluoroscopic examination in pleurisy, 476.

Follicles at base of tongue, enlargement of, in status lymphaticus, 547.

Fomentations, hot, in multiple neuritis, 663; in acute nephritis, 579; in pleurisy, 479.

Fontanelles, closure of, 18.

Food, carbohydrates in, 103; fat in, 100; mineral salts in, 104; percentages, 139; poisoning from, a cause of intestinal disorders, 176; proteins in, 101; relation of, to weight, 14; water in, 105.

Food idiosyncrasies, a cause of eczema, 719, 722; a cause of urticaria, 726.

"Food injuries," intestinal indigestion from, 184, 185; in recurrent vomiting, 252.

Foods, additional, in infancy and early childhood, 142, 143; percentage value of, 139; proprietary, 127; value of different artificial, 118.

"Foot-drop," 662.

Forchheimer, on accentuation of second sound in diagnosis of heart disease, 508; on adenitis in scarlet fever, 331; on alkali treatment of rheumatism decreasing risk of endocarditis, 492; on acute cardiac dilatation due to coughing spells in influenza, 496; on chlorate of potash in treatment of stomatitis ulcerosa, 151; on enanthem of rubella, 342; on enlargement of muciparous follicles in stomatitis catarrhalis, 147; on use of flexible collodion in adenitis, 544; on fungi present in stomatitis mycosa, 148; on heat as cause of gastroenteric intoxication, 180; on heatstroke as a cause of fever in infancy, 262; on heredity in etiology of appendicitis, 223; on high enemata in typhoid fever for relief of meteorism, 278; prescription by, for stimulating remedy, 196; on splanchnic paralysis following diphtheria, 307; on therapeutics of chlorosis, 528; on transient systolic murmur in myocarditis, 507.

Foreign bodies in larynx, trachea, and bronchi, 435; prognosis in, 437; use of X-ray in, 436.

Formalin vapor in treatment of whooping-cough, 293.

Formaldehyde, as disinfectant after contagious diseases, 328, 339; method of generating, 328.

Fowler's solution of arsenic, use of, after removal of neoplasms from larynx, 435; in malaria, 286; in purpura, 536; in chronic tuberculosis, 400.

Frankel's diplococcus pneumoniæ, 442.

Freeman on temperature of pasteurized milk, 120.

Freeman pasteurizer, 121.

Freidreich's disease. *See Hereditary ataxia.*

Fresh air, necessity of, in infancy and childhood, 134; necessity of, to new-

born, 4; therapeutic value of, 43; in acetonuria, 567; in bronchopneumonia, 464, 465; in lobar pneumonia, 450; in acute nephritis, 581; in pleurisy, 478; in pseudoleukemia, 526; in purpura, 535; in rickets, 244; in status lymphaticus, 550; in tuberculosis, 401.

FRIEDLÄNDER, A., experiments by, on effect of X-ray treatment on thymus gland, 549; on lymphocytosis in tuberculosis of lymph-nodes, 383; on urotropin in treatment of mumps, 360.

FRIGHT, a cause of chorea, 698.

FRONTAL SINUSES, brain abscess from septic infection of, 618; infection of, in influenza, 312; purulent infection of, in diphtheria, 300.

FURUNCULOSIS, 728; microorganisms present in, 728; prognosis of, 729; symptomatology of, 728; treatment of, 729; in typhoid fever, 271, 274; vaccine therapy for, 58, 729.

G

GAS BACILLUS. *See Bacillus Welchii.*

GASTRIC INDIGESTION, ACUTE, 158; etiology of, 158; improper food a cause of, 159; physiological gastric incompetency, 158; prognosis, 159; symptomatology, 159; treatment, 160.

GASTRIC CATARRH. *See Chronic gastritis.*

GASTRITIS, ACUTE, 160; corrosive form of, 161, 163; etiology of, 160; noncorrosive form of, 160, 162; pathology of, 160; symptomatology of, 162; temperature changes in, 161; treatment of, 162.

GASTRITIS, CHRONIC, 170; diagnosis of, 171; diet in, 172; etiology of, 170; hygiene in, 173; pathology of, 171; prognosis of, 172; stomach washing in, 172; symptomatology of, 171; treatment of, 172.

GASTROENTERITIS, after bronchopneumonia, 462; in influenza, 310, 314.

GASTRODUODENITIS, 165.

GASTROINTESTINAL DISORDERS, albumin water of value in, 128; bronchopneumonia associated with, in new-born, 461; buttermilk of value in, 123; chorea caused by, 697; drugs in causation of, 39; eczema due to, 719; epilepsy associated with, 695; Finkelstein's albumin milk of value in, 123; frequency of, in congenital heart disease, 489; fresh air of value in, 44; headache due to, 709; indicanuria present in, 562; insomnia associated with, 676, 677; meat preparations of value in, 129; mortality from, in infancy, 2; Nestlé's food of value in, 127; pavor nocturnus associated with, 671; premature infants susceptible to, 64; rickets associated with, 240; skimmed milk of value in, 124; urticaria due to, 726; water of value in, 47, 128.

GAUCHER, on primary splenomegaly, 552.

GAVAGE, indications for, 50.

GAY AND LUCAS, on blood changes in acute anterior poliomyelitis, 649.

GELATIN, subcutaneous injection of, in hemophilia, 539; in hemorrhage of new-born, 83.

GEOGRAPHICAL TONGUE, 155.

GERHARDT, on physical signs in persistent patulous ductus arteriosus, 488.

GERMAN MEASLES. *See Rubella.*

GIBSON, on diagnostic value of discharge of bloody mucus in intestinal intussusception, 220.

GIGANTOBLASTS, 515.

GLOSSITIS, DESQUAMATIVE. *See Geographical tongue.*

GLYCERIN OINTMENT, use of, in acute catarrhal bronchitis, 441; in mumps, 360.

GOLDBERG AND ANDERSON, on production of measles in monkeys, 332.

GONOCOCCUS OF NEISSER, in cystopyelitis, 584; in vulvovaginitis, 591, 592, 595.

GONOCOCCUS VACCINE, therapeutic use of, 58.

GOUT, a cause of asthma, 711; of eczema, 722; of headache, 708.

GRAND MAL, 692.

GRIFFITH, CROZER, on diagnosis between appendicitis and lobar pneumonia, 449; on incubation period of rubella, 341; on prognosis in typhoid fever, 275.

GROZY, experiments by, on dextrimaltose preparations, 125.

GRUBER-WIDAL REACTION, in paratyphoid fever, 275; in typhoid fever, 265, 269, 273; in diagnosis between typhoid fever and tuberculous peritonitis, 391.

GRUNBAUM, on typhoid fever in the chimpanzee, 267.

GUAIACOL, therapeutic use of, in acute catarrhal bronchitis, 441; in convalescence from bronchopneumonia, 469; in cystopyelitis, 587; in measles, 340; in pseudoleukemia, 526; in tuberculous peritonitis, 401; in typhoid fever, 277; in urticaria, 728.

GUAIACOL, inhalations of, in acute laryngitis, 433; in acute rhinitis, 414.
GUAIACOL OINTMENT, use of, in adenitis, 544; in acute catarrhal bronchitis, 442; in bronchopneumonia, 469; in cystopyelitis, 587; in measles, 340; in mumps, 360; in tuberculosis, 398; in tuberculous bronchopneumonia, 401; in tuberculous peritonitis, 401.
GUANIER, description by, of microorganisms in variola, 345.
GUMMATOUS ULCERATION, in late hereditary syphilis, 367.
GYMNASTIC EXERCISES, therapeutic value of, 53; value of, in rachitis, 245.
"GYROSPASM," 689.

H

HABIT, a cause of enuresis, 600; a cause of pseudomasturbation, 606.
HABIT SPASM, 701; definition of, 701; etiology of, 702; prognosis of, 702; symptomatology of, 702; treatment of, 703.
HADDEN, W. B., on head-nodding in child, 689, 690.
HALL, on value of pressure signs in lymph node tuberculosis, 385.
HAMBURGER, description of skin tuberculides, by, 384.
HAMILL, on late systolic pulmonary murmur, 507; on percussion, 32, 33.
HAMILTON, ALICE, on susceptibility of vulvovaginal canal in young children to gonorrheal infection during scarlet fever, 591; on vaccine therapy in treatment of gonorrheal vaginitis, 594.
HARDAWAY AND GRINDON, prescription by, for ointment for use in eczema, 724; for ointment for use in tinea tonsurans, 734; for lotion for use in urticaria, 727; on Crocker's method of treatment in tinea tonsurans, 735.
HARELIP, 156.
HASTINGS, measurements by, of growth in children, collected by, 17.
HEAD AND NECK, examination of, in illness, 30.
HEAD, measurements of, in childhood, 18; nodding, 689; palpation of, 30; position of, in illness, significant, 29.
HEADACHE, 708; anemic form of, 708; due to brain tumor, 616; etiology of, 708; in meningococcic meningitis, 632; organic form of, 709; reflex form of, 709; remedies for relief of, 710; toxic forms of, 709; treatment of, 709; due to uremia, 576.
HEARING, sense of, in infants, 22.
HEART, acceleration of action in infancy, 483; displacement of, in pleurisy, with effusion, 473, 476; disturbance of, in acute articular rheumatism, 404; disturbance of, in chorea, 700; enlargement of, in congenital heart disease, 486.
HEART, disease of, associated with acute articular rheumatism, 404; displacement of, in pleurisy with effusion, 473, 476; disturbance of, in chorea, 700; enlargement of, in stenosis of pulmonary artery, 487; growth of, in infancy and childhood, 484; hypertrophy of, in persistent patulous ductus arteriosus, 488; murmurs, *see Murmur;* peculiarities of, in childhood, 483; rest, in treatment of disease of, 504.
HEART, ACUTE DILATATION OF, 496; diagnosis of, 496; diagnosis of, from pericarditis, 511; etiology of, 496; prognosis of, 496; prophylaxis of, 497; treatment of, 498.
HEART DISEASE, CONGENITAL, 484; blood changes in, 486; differential diagnosis between different forms of, 486; etiology of, 484; malformations a cause of, 485; special lesions in, 486; symptomatology of, 485; treatment of, 488.
HEART DISEASE, FUNCTIONAL, 506; arrhythmia, as form of, 506; bradycardia, as form of, 507; diet in, 508; etiology of, 506; frequency of, in child, 506; murmurs associated with, 507, 508; paroxysmal tachycardia, as form of, 506; treatment of, 508.
HEART, CHRONIC VALVULAR DISEASE OF, 498; acute articular rheumatism, a cause of, 498; aortic regurgitation a form of, 501; aortic stenosis a form of, 501; compensation in, 502; diet in treatment of, 504; drugs in treatment of, 504; endocarditis in causation of, 498; etiology of, 498; exercise in treatment of, 504; failure of compensation in, 502; hygiene in treatment of, 504; mitral regurgitation of, 499; mitral stenosis a form of, 500; prognosis in, 502; treatment of, 503; treatment of failing compensation in, 505; tricuspid regurgitation, 502.
HEAT-DISSIPATING MECHANISM, increasing stability of, 27; relative importance of, in child, 26.
HEAT, INHIBITORY CENTERS. *See Thermoinhibitory centers.*

HEAT-PRODUCING CENTERS. *See Thermogenic centers.*
HEAT-REGULATING MECHANISM, development of, 25; peculiarities of, in premature infant, 62.
HEAT STROKE, 262.
HEBRA'S FORMULA, 725.
HEIDINGSFELD, formula for lotion by, 724; formula for ointment by, 735; on treatment of pediculosis capillitii, 737.
HEIGHT, AT DIFFERENT AGES, 17.
HEIMANN, on demonstration of gonococcus in gonorrheal vaginitis, 592.
HEKTOEN, on relation of streptococcemia to severity of scarlet fever, 317.
HEMATOMA OF STERNOCLEIDOMASTOID MUSCLE IN NEW-BORN, 98.
HEMATURIA, 562; in acute nephritis, 577; in tumor of the kidney, 588; treatment of, 563.
HEMIPLEGIA, 612; treatment of, 615.
HEMOGLOBIN, variations of, in blood of infants, 519; in chlorosis, 526; in Hodgkin's disease, 541; in pernicious anemia, 529; in pseudoleukemia, 525; in simple secondary anemia, 523; in status lymphaticus, 547.
HEMOGLOBINURIA, 563; diagnosis between, and hematuria, 564; epidemic form of, in new-born, 82, 564; etiology of, 563; paroxysmal, 564; due to poisons, 564; prognosis of, 565; treatment of, 565; types of, 564.
HEMOPHILIA, 537; definition of, 537; diagnosis of, 539; drugs in treatment of, 539; etiology of, 537; prophylaxis of, 539; serum treatment of, 540; symptomatology of, 538; treatment of, 539.
HEMORRHAGE, cerebral, a cause of mental deficiency, 623; from conjunctiva in whooping-cough, 289; meningeal, a cause of infantile palsy, 610; from mucous membranes in purpura, 533; from the respiratory tract in whooping-cough, 289; in scurvy, 248; in typhoid fever, from intestines, 272, 278.
HEMORRHAGE IN NEW-BORN, 82; due to congenital syphilis, 83; due to epidemic hemoglobinuria, 82; etiology of, 82; due to acute fatty degeneration, 82; from intestines, 4; due to melena neonatorum, 82; prognosis of, 83; due to septic infection, 78; transfusion for relief of, 84; treatment of, 83; from umbilicus, 85.
HENOCH, on duration of typhoid fever, 269; on pneumonia complicating measles, 337; on a rare form of non-tuberculous peritonitis, 391.
HENOCH'S PURPURA, 534; arsenic contraindicated in, 537.
HEREDITY, influence of, in causation of adenoid disease, 425; of appendicitis, 223; of acute articular rheumatism, 402; of asthma, 711; of congenital heart disease, 485; of congenital ichthyosis, 730; of eclampsia, 679; of eczema, 719; of acute endocarditis, 489; of epilepsy, 691; of habit spasm, 702; of headache, 708; of hemophilia, 537; of hereditary ataxia, 657; of hydrocephalus, 619; of hysteria, 705; of idiocy, 623; of insomnia, 676; of masturbation, 606; of nystagmus, 690; of orthostatic albuminuria, 569; in over-fatigue at school, 9; of pavor nocturnus, 671; of progressive muscular dystrophy, 666; of pseudomasturbation, 606; of recurrent vomiting, 252; of sporadic cretinism, 553; of tuberculosis, 375; of urticaria, 726.
HERNIA, associated with rickets, 240; caused by whooping-cough, 289; congenital, 87; umbilical, in new-born, 86; umbilical, in sporadic cretinism, 554.
HEROIN HYDROCHLORATE, use of, in influenza, 314; in whooping-cough, 294.
HERTER, on indolaceturia, 562; on results of acid intoxication, 566.
HEUBNER, on food consumption in infants, 131; on immunity of young infants to scarlet fever, 315; on orthostatic albuminuria, 568, 569.
HEXAMETHYLENAMIN. *See Urotropin.*
HIP-JOINT, tuberculosis of, 392.
HIRSHSPRUNG, description by, of congenital dilatation of colon, 208.
HISTORY-TAKING IN ILLNESS, 28.
HOCHSINGER, on peculiarities of pericarditis in children, 509; on physical signs in defective septum, 488; on relation between heart weight and body weight in infancy, 483; on syphilitic pseudoparalysis, 365; on X-ray treatment of enlarged thymus, 549.
HODGE, C. F., on changes in nerve cells as result of nervous activity, 11.
HODGKIN'S DISEASE, 540; blood changes in, 541; definition of, 540; diagnosis of, 541; etiology of, 540; pathology of, 540; prognosis of, 542; symptomatology of, 541; temperature changes in, 541; treatment of, 542.
HOFFMANN, gastrostomy performed by,

in congenital malformation of the esophagus, 158.
Hoffmann's sign, 688.
Holding-the-breath spells, in laryngismus stridulus, 685.
Holt, apparatus devised by, for examining breast milk, 110; on use of atropin in enteric infection, 195; on use of atropin in treatment of enuresis, 602; on composition of human milk, 110; on dilatation of stomach, 163; on gonococcus in vulvovaginitis, 591; on method of obtaining mucus for examination in tuberculosis, 389; on use of morphin in enteric infection, 195; on mortality in measles, 338; on primary cases of nephritis, 575; on quantity of milk required by normal infant, 110; report by, on gonorrheal arthritis, 593; on salicylate of soda to control gastric fermentation, 173; on skimmed milk in diet of infants, 126; on transmission of cancrum oris, 152.
Holt's inanition fever, 87, 263; diagnosis of, 88; etiology of, 88; obscure rise of temperature due to, 263; symptomatology of, 88; treatment of, 89.
Holt and Bartlett, on acute anterior poliomyelitis, 642, 650.
Holt and Northrup, on primary intestinal tuberculosis, 380.
Hookworm, presence of, in pernicious anemia, 528, 529.
Howlands and Richards, on recurrent vomiting, 251, 254.
Huber, analysis by, of cases of meningococcic meningitis, 632; of blood examination in meningococcic meningitis, 634.
Human milk. *See Milk.*
Hutchinson, Jonathan, formula by, for use in tinea tonsurans, 734; on late hereditary syphilis, 366.
Hutchinson's teeth, 146.
Hyacin hydrobromate, in chorea, 701.
Hydrencephalocele, 622.
Hydriodic acid, syrup of. *See Syrup.*
Hydrocele, 597.
Hydrocephalus, acute, 618.
Hydrocephalus, chronic internal, 618; definition of, 618; etiology of, 619; imbecility associated with, 623; mortality from, 620; pathology of, 619; prognosis of, 620; symptomatology of, 619; treatment of, 620.
Hydrochloric acid, therapeutic use of, in typhoid fever, 277.
Hydronephrosis, 589.
Hydrotherapy, 44; use of, in hysteria, 708; in lobar pneumonia, 451; in acute nephritis, 579; in purpura, 536; in chronic rheumatic arthritis, 409; in scarlet fever, 329; in typhoid fever, 277; value of, as means of treatment, 44.
Hygiene, general, principles of, in infancy and childhood, 2.
Hygienic treatment, of bronchopneumonia, 465; of enteric infection, 197; of chronic intestinal indigestion in infants, 201; of lobar pneumonia, 450; of recurrent vomiting, 257.
Hymenolepis nana, 212.
Hypertrophy of heart, in persistent patulous ductus arteriosis, 488; in chronic valvular heart disease, 499.
Hypertrophy of the pyloris, congenital. *See Congenital.*
Hypodermoclysis, 48; use of, in diphtheria, 307; method of using, 48; use of, in nephritis, 580; use of, in scarlet fever, 330; therapeutic value of, 48; use of, in typhoid fever, 278.
Hypospadias, 597.
Hysteria, 706; treatment of, 707.

## I

Ice, therapeutic use of, 47.
Ice-bag, therapeutic use of, in adenitis of scarlet fever, 331; in acute cardiac dilatation, 498; in cystopyelitis, 587; in diphtheria, 304; in acute endocarditis, 493; in fevers, 266; in headache, 701; in heart complications of acute rheumatism, 406; in Henoch's purpura, 537; in intestinal hemorrhage, 278; in malaria, 286; in mastoiditis, 718; in meningococcic meningitis, 638; in acute pericarditis, 512; in pleurisy, 478; in purpura hemorrhagica, 537; in tonsillitis, 423; in typhoid fever, in connection with baths, 277, 278; in variola, 348.
Ice-cap, therapeutic value of, 46; in eclampsia, 682; in lobar pneumonia, 451.
Ice-cream, a source of infection in typhoid fever, 268.
Ichthyol ointment, therapeutic use of, in erysipelas of new-born, 81; in scarlet fever, 330.
Ichthyosis, congenital. *See Congenital.*
Icterus neonatorum, 92; symptomatology of, 93; treatment of, 93.
Icterus, other forms of, in new-born, 94.

Icterus, in toxic nephritis, 572; in Winckel's disease, 564.
Idiocy, 622; acquired, 623; amaurotic family, 624; congenital, 622; definition of, 622; due to epilepsy, 694; etiology of, 622; microcephalic, 625, 626; Mongolian, 554, 625; prognosis of, 624; sporadic cretinism, 554; symptomatology of, 623; treatment of, 624.
Iglauer, on avoidance of general anesthesia in evacuation of retropharyngeal abscess, 429.
Imbecility, associated with epilepsy, 694; with infantile cerebral palsies, 613, 614.
Impetigo contagiosa, 731; treatment of, 732.
Inanition fever, Holt's. *See Holt.*
Incontinence of urine, occurrence of, in eclampsia, 681; in hypospadias, 598; in myelitis, 654, 656; in night-terror, 673; physiologically in infancy, 561.
Incubator, 66.
Indican, absence of, in urine of new-born, 562; derivation of, in urine, 562.
Indicanuria, 562; in headache, 709; in chronic intestinal indigestion, 202; in intestinal toxemia, 265; in orthostatic albuminuria, 569; a sign of putrefactive changes in intestine, 205.
Indolaceturia, cause of, 562; presence of, in intestinal toxemia, 205; presence of, associated with putrefactive changes of intestine, 205.
Infancy, chief causes of death in, 1; clothing in, 5; contagion in, 6; fresh air in, 4; head-measurements in, 18; heat-regulating mechanism in, 25; height in, 17; hygiene during, 2, 10; malaria in, 283; muscular development in, 20; nervous activity in, 6; nervous system in, 23; nursery in, 5; peculiarities of blood in, 518; rest in, 4; sleep in, 4, 671; special senses in, 22; spinal development in, 22; susceptibility to high temperatures in, 27, 260; therapeutics of, 38; weight in, 13, 15.
Infantile atrophy, 199.
Infantile cerebral palsies, 610; definition of, 610; diagnosis of, 615; diet in, 615; etiology of, 610; orthopedic treatment of, 616; pathology of, 611; prognosis of, 615; symptomatology of, 612; treatment of, 615.
Infantile myxedema. *See Cretinism, sporadic.*
Infantile spinal paralysis. *See Acute anterior poliomyelitis.*
Infantile scurvy, 246; blood changes in, 248; curative treatment of, 249; definition of, 246; diagnosis of, 248; diet of, 249; etiology of, 246; morbid anatomy in, 247; prognosis of, 249; prophylactic treatment of, 249; rheumatism mistaken for, 404; symptomatology of, 247; temperature changes in, 248; toxic nephritis associated with, 572; treatment of, 249; urine findings in, 248.
Infection, sources of, in adenitis, 377; in adenoid disease, 425; in appendicitis, 223; in acute catarrhal bronchitis, 438; in diphtheria, 295; in acute endocarditis, 490; in enteric infection, 189; in erythema infectiosum, 343; in gonorrheal vulvovaginitis, 591; in impetigo contagiosa, 731; in influenza, 308; in acute intestinal toxemia, 176; in lobar pneumonia, 433; in malaria, 280; in mastoiditis, 717; in measles, 332; in meningococcic meningitis, 631; in mumps, 357; in mycotic nephritis, 574; in myelitis, 652; in myocarditis, 494; in acute nephritis, 572; in chronic nephritis, 584; in ophthalmia neonatorum, 94; in otitis media, 714; in pericarditis, 509; in peritonitis, 228, 229; in acute anterior poliomyelitis, 642; in retropharyngeal abscess, 427; in acute rhinitis, 411; in rubella, 341; in scarlet fever, 316; in sepsis of new-born, 75; in sinuses, frontal and ethmoid, from diphtheria, 300; in syphilis, 361; in tetanus neonatorum, 89; in tonsillitis, 419; in tuberculosis, 373; in tuberculous meningitis, 627; in typhoid fever, 268; in simple vaginitis, 595; in variola, 345.
Infection, susceptibility to, in premature infant, 64.
Infectious diseases, acute, chorea following, 697; congenital heart disease due to, in mother, 485; cystopyelitis due to, 584; danger from, in status lymphaticus, 550; eclampsia due to, 680; endocarditis due to, 490; enuresis due to, 599; milk of nursing mother affected by, 112; acute nephritis due to, 578; mycotic nephritis due to, 574, 575; predisposing cause in gonorrheal infection of vagina, 595; predisposing cause in transmission of tuberculosis, 375; purpura due

to, 532; splenic enlargement associated with, 552; danger from, in status lymphaticus, 550; urine examination in, 559.

INFLUENZA, 308; after-treatment of, 315; bacillus of, 308, 311; acute cardiac dilatation due to, 496; characteristics of, in infancy, 311; chronic form of, 311; climatic influences in, 308; climatic treatment of, 310, 314; complications in, 311; coryza in, 310; definition of, 308; danger from, in infancy, 5; danger from, to tuberculous children, 595; definition of, 308; diet in, 313; drugs in, 313; etiology of, 308; eruptions in, resembling scarlet fever, 325; gastroenteritis in, 310, 311; immunity from, 311; incubation period in, 309; local treatment of, 314; mortality from, in infancy, 2; multiple neuritis from, 661; nephritis due to, 312; nervous symptoms in, 308; pathology of, 309; prognosis of, 312; prophylaxis of, 312; nervous symptoms in, 308; pathology of, 309; prognosis of, 312; prophylaxis of, 312; skin eruptions in, 310, 311; sources of infection in, 308; symptomatology of, 309; symptom groups in, 310; temperature curve in, 309, 311; treatment of, 313; eruptions in, 310, 311; sources of infection in, 308; symptomatology of, 309; symptom groups in, 310; temperature curve in, 309, 311; treatment of, 313.

INHALATIONS, in treatment of acute laryngitis, 433; of laryngeal diphtheria, 304; of acute rhinitis, 433.

INHIBITORY FUNCTION, development of, 24.

INJECTIONS, HYPODERMIC, of adrenalin, 307; of caffein, 307, 330; of camphor, 307; of ether, 307; of salt solution, 580; therapeutic value of; *see Hypodermoclysis.*

INJECTIONS, INTRAVENOUS, in acute nephritis, 579.

INJECTIONS, NASAL, in epistaxis, of adrenalin, 416; in acute rhinitis, 414.

INJECTIONS, RECTAL, of Fischer's alkaline solution in acute nephritis, 579, 580; of food in artificial feeding, 52; of olive oil in constipation, 51; of salt water in constipation, 51, 207; of salt water in flushing the colon, 51; therapeutic value of, 51.

INJECTIONS, VAGINAL, in gonorrheal vulvovaginitis, 594.

INOCULATION, ANTI-TYPHOID, 276.

INORGANIC CONSTITUENTS OF MILK, 104; necessity of, to nutrition, 105.

INSOLATION. *See Heat-stroke.*

INSPECTION, EXAMINATION BY, IN SICKNESS, 29; of face, 29; of general position, 30; of head, 29; of respiration, 30; of skin, 30.

INTESTINAL COLIC, 184.

INTESTINAL DISCHARGES, examination of, in illness, 34; danger in handling, 179; nature of, in intussusception, 220.

INTESTINAL DISORDERS OF INFANCY, 173; age a predisposing cause, 179; bad hygiene a predisposing cause, 180; bacteria present in, 176, 177, 178, 179; bathing in prophylaxis of, 182; change of climate in treatment of, 181; clothing in prevention of, 183; dentition as a cause of, 176; etiology of, 173; exposure to cold and wet a cause of, 176; over-feeding a cause of, 175; predisposing factors in, 174; prophylaxis of, 180; summer heat a predisposing factor in, 179; swallowing mucus a cause of, 176.

INTESTINAL FERMENTATION, 103.

INTESTINAL INDIGESTION, ACUTE, diagnosis of, 187; diarrhea in, 183; fat indigestion in, 185; food injuries in, 184, 185; nervous symptoms in, 184; pain in, 184; pathology of, 183; prognosis of, 187; protein indigestion in, 186; sugar indigestion in, 186; symptomatology of, 183; symptom group in, 184; temperature curve in, 184; treatment of, 187.

INTESTINAL INDIGESTION IN INFANTS, CHRONIC, 197; diet in, 200; etiology of, 197; hygienic treatment of, 201; infantile atrophy from, 199; marasmus in, 200; medicinal treatment of, 201; pathology of, 198; symptomatology of, 198; treatment of, 200.

INTESTINAL INDIGESTION IN OLDER CHILDREN, CHRONIC, 201; climate in treatment of, 203; diet in, 203; etiology of, 201; medicinal treatment of, 203; prognosis of, 203; symptomatology of, 202; treatment of, 203.

INTESTINAL INTOXICATION. *See Intestinal toxemia.*

INTESTINAL OBSTRUCTION, ACUTE, in appendicitis, 224.

INTESTINAL PARASITES. *See Parasites.*

INTESTINAL PERFORATION, in typhoid fever, 272, 279.

INTESTINAL TOXEMIA, 176; a cause of

asthma, 711; of eclampsia, 680; of fever, 261; of headache, 709.
Intestinal tuberculosis, 380.
Intubation, in acute laryngitis, 433; in edema of the larynx, 434; in laryngeal diphtheria, 304, 305; method of performing, 305.
Intussusception of the intestines, 219; course of, 221; definition of, 219; diagnosis of, 222; etiology of, 219; intestinal discharges in, 220; pathology in, 220; physical signs of, 221; prognosis of, 221; symptomatology of, 220; temperature curve in, 221; treatment of, 221; vomiting in, 220.
Inunctions of guaiacol, therapeutic use of, in adenitis, 544; in bronchopneumonia, 469; in acute catarrhal bronchitis, 442; in lobar pneumonia, 452; in measles, 340; in pleurisy, 479; in tuberculous bronchitis, 401.
Inunctions, drugs suitable for use by, 42, 43; method of giving, 42; of oil in rickets, 245; in scarlet fever, 330; therapeutic value of, 41.
"Inward spasm." *See Laryngismus stridulus.*
Iodid of potash. *See Potassium iodid.*
Iodid of iron, syrup of. *See Syrup.*
Iodin, use of, by inunction, 42, 43; in chronic pericarditis, 513; in pleurisy, 479; in tuberculosis of lymph-nodes, 399.
Iodin, therapeutic use of internally, in adenitis, 544; in sporadic cretinism, 556; in syphilis, 371; in tuberculosis, 399.
Iodid of potassium. *See Potassium iodid.*
Ipecac, syrup of. *See Syrup.*
Iron, importance of, to nutrition, 104; tincture of chlorid of, 81.
Iron, syrup of iodid of. *See Syrup.*
Iron, therapeutic use of, in adenitis, 544; in asthma, 713; in acute cardiac dilatation, 498; in chlorosis, 527, 528; in chorea, 701; in convalescence from influenza, 315; in convalescence from acute laryngitis, 433; in convalescence from lobar pneumonia, 451; in enuresis, 601; in facial paralysis, 666; in hemophilia, 539; in multiple neuritis, 664; in nystagmus and head-nodding, 691; in pavor nocturnus, 675; in prevention of laryngismus stridulus, 686; in pseudoleukemia, 526; in pseudomasturbation, 609; in purpura, 536, 537; in rickets, 245; in simple anemia, 524; in status lymphaticus, 580; in tonsillitis, 423; in tetany, 689; in valvular heart disease, 505.
Irrigation, of bladder in cystopyelitis, 587; of colon in enteric infection, 51, 193, 195; of vagina in gonorrheal vulvovaginitis, 594; of stomach; *see Gavage.*
Insomnia, 675; etiology of, 676; prophylaxis of, 677; treatment of, 617; varieties of, 676.
Isolation, in influenza, 312; in measles, 338; in mumps, 360; in scarlet fever, 327; in tonsillitis, 422; in typhoid fever, 276; in variola, 348.
Itch. *See Scabies.*

J

Jacksonian epilepsy, 693.
Jacobi, on use of belladonna in whooping cough, 293; on use of cereals in artificial feeding, 126; on diet in fevers, 266; on lymphoid ring of pharynx as portal of infection, 490; on mitral stenosis without a heart murmur, 500; on peroxid of hydrogen as an irritant to the throat, 423; on rickets as cause of laryngismus stridulus, 684; on relative length of colon in infectious disease, 204; on strapping chest for relief of pain in pleurisy, 478; on tonsillitis, as opening for infection, 478; on uric acid in infants, 561.
James, apparatus devised by, for breathing exercises after operation for empyema, 481.
Jaundice, catarrhal form of; *see Gastroduodenitis;* of new-born; *see Icterus neonatorum;* in septic infection of new-born, 77.
Jenner, Edward, discovery of vaccination by, 349.
Jehle, on lordosis of lumbar vertebræ, as cause of orthostatic albuminuria, 569.
Johnson, W. W., on mortality from whooping-cough, 291.
Joints, tuberculosis of, 381, 391; treatment of, 401.
Joint contraction in hysteria, 706.

K

Kassowitz and Jacobi, on phosphorus in treatment of rickets, 245.
Keller, on injury to metabolic processes

from over-feeding, 175; on intestinal disorders following over-feeding, 174, 175; introduction by, of malt soups as artificial food for infants, 124; on symptom group in acute intestinal indigestion, 184.

KERATITIS, SYPHILITIC INTERSTITIAL, 367.

KERLEY, on care of milk in tenements, 182; on composition of human milk, 108; on gavage, 50; on method for preventing pseudomasturbation, 182; on rectal enemata of olive oil in constipation, 51, 207.

KERNIG'S SIGN, significance of, in sick child, 32; significance of, in meningococcic meningitis, 633; in acute anterior poliomyelitis, 645; in purulent meningitis, 633, 635; in tuberculous meningitis, 628.

KIDNEY, cystic degeneration of, 589; dislocation of, 590; enlargement of, in syphilis, 359, 366; size of, at birth, 590; tuberculosis of, 590.

KIDNEY TUMORS, 587; etiology of, 587; mortality from, 589; nature of, 587; operative treatment for, 589; symptomatology of, 588; treatment of, 589.

KILMER, on use of abdominal belt in whooping cough, 293.

KIRMISSON, operation by, for obstruction of esophagus, 158.

KJELBERG, on gastrointestinal disease, as cause of toxic nephritis in infancy, 572.

KLEBS-LÖFFLER BACILLUS, description of, 295; in diagnosis between diphtheria and tonsillitis, 420; in etiology of diphtheria, 295; in stomatitis gangrenosa, 153.

KLEIN AND GORDON, description by, of streptococcus scarlatina, 317.

KNEE-JOINT TUBERCULOSIS, 393.

KOCH'S BACILLUS, 273.

KOCH'S TUBERCULIN, therapeutic use of, 58.

KOLMER AND WESTON, on vaccine therapy in sequelæ of scarlet fever, 332.

KÖNIG, on results of removal of thymus gland, 551.

KOPLIK, on diagnosis of hypertrophy of pylorus, 167, 168; on diagnosis of pyloric spasm, 168; on enanthem stage of measles, 334; on frequency of simple vulvovaginitis in children, 595; on gonococcus peritonitis, 230; on gastrointestinal disease as cause of toxic nephritis, 572; on inflammation of cervix uteri in gonococcus vulvovaginitis, 592; on presence of pneumococcus in pleurisy in children, 470; on presence of streptococcus in retropharyngeal abscess, 248; tables by, on physical measurements of average boys and girls, 17, 18; on temperature of milk in infant feeding, 120.

KURTH, description by, of streptococcus conglomeratus, 317.

KÜSTNER, OTTO, on rudimentary condition of genitourinary organs in little girls, 604.

KOPLIK SPOTS, 335.

## L

LADD AND WETTER, on influence of antimeningitic serum on joint affection, 635.

LA FÉTRA, on eosinophilia in asthma.

LANDOUZY-DÉJERINE TYPE OF PROGRESSIVE MUSCULAR DYSTROPHY, 668.

LANDRY'S PARALYSIS, 645.

LANDSTEINER AND POPPER, on inoculation of monkeys with acute anterior poliomyelitis, 641.

LANGSTEIN, on symptom group in acute intestinal indigestion, 184.

LANOLIN, therapeutic use of, by inunction, 43; in measles, 339; in acute rhinitis, 415; in rickets, 245; in scarlet fever, 330; in varicella, 359.

LARYNGEAL STRIDOR, 437; treatment of, 438.

LARYNGISMUS STRIDULUS, 684; definition of, 684; diet in, 686; etiology of, 684; prevention of attack, 685; prognosis of, 685; symptomatology of, 685; treatment of attack, 685; treatment of underlying condition, 686.

LARYNGITIS, ACUTE, 429; definition of, 429; diagnosis of, 431; drugs in treatment of, 432; dyspnea in, 431; etiology of, 429; microorganisms present in, 429; pathology of, 430; prognosis of, 432; sources of infection in, 429; spasmodic form of, 430; symptomatology of, 430; temperature curve in, 431; treatment of, during attack, 432; treatment of, during interval, 433.

LARYNGITIS, catarrhal form of, in measles, 337; membranous form of, in measles, 337, 340; syphilitic form of, in new-born, 364.

LARYNGOSPASM. *See Laryngismus stridulus.*

Larynx, diseases of, 429; congenital laryngeal stridor, 437; edema of the larynx, 434; foreign bodies in, 435; acute laryngitis, 429; neoplasms in, 435.
Lavage, 49; in chronic gastritis, 172; in hypertrophy of the pylorus, 170.
Laveran, discovery by, of plasmodium malariæ, 279.
Lead poisoning, a cause of multiple neuritis, 661.
le Boutillier, on late systolic pulmonary murmur, 507.
Ledermann, on Wassermann reaction in hereditary syphilis, 368.
Leeches, therapeutic use of, in acute cardiac dilatation, 498; in mastoiditis, 718.
Leukemia, 530; blood picture in, 530; definition of, 530; diagnosis between, and Hodgkin's disease, 542; diagnosis between, and pseudoleukemia, 525; etiology of, 530; lymphoid form of, 530; myeloid form of, 530; prognosis of, 531; symptomatology of, 530; temperature changes in, 531; treatment of, 531.
Leukocytes. *See Blood corpuscles, white.*
Leukocytosis, in chlorosis, 527; in diagnosis of sepsis, 265; in diagnosis of typhoid fever, 273; in diphtheria, 297; in infancy, 519; in influenza, 310; in lobar pneumonia, 447; in measles, 336; in meningococcic meningitis, 634; in mumps, 359; in poliomyelitis, 649; in rubella, 343; in simple anemia, 523; in status lymphocytosis, 547; in syphilis, 366; in whooping cough, 289; in varicella, 356; in variola, 347.
Leveran and Catrin, investigation by, of blood in mumps, 357.
Ligaments, weakness of, in rickets, 240.
Lime water, value of, in artificial feeding, 139.
Lipase, presence of, in milk, 106.
Lithuria, 561.
Liver, abscess of, in lobar pneumonia, 448; displacement of, in pleurisy with effusion, 476; enlargement of, in simple anemia, 523; enlargement of, in pseudoleukemia, 531; functional disturbance of, in purpura, 532; incompetency of, a cause of recurrent vomiting, 251.
Löffler, on blood serum, 295; on children as carriers of malarial parasites, 281.
Longstein, on depreciation of health in orthostatic albuminuria, 569.
Lord, on leukocytosis in influenza, 310.
Lovett, table by, showing increase in epidemic form of acute anterior poliomyelitis.
Lumbar puncture, diagnostic value of, 37; in differentiating the types of meningitis, 640; in meningococcic meningitis, 634; method of, 27; in acute anterior poliomyelitis, 651; in tuberculous meningitis, 630.
Lungs, enlargement of, in syphilis, 362; tuberculosis of; *see Pulmonary tuberculosis.*
Lussatti and Biolchini, isolation of fat-splitting ferment by, 106.
Lüthje, on frequency of late systolic pulmonary murmur, 507.
Lymph-adenoma. *See Hodgkin's disease.*
Lymph nodes, enlargement of, in adenitis, 543; in adenoid disease, 543; in diphtheria, 296; in Hodgkin's disease, 540, 541; in kidney tumors, 588; in leukemia, 530; in mumps, 359; in retropharyngeal abscess, 428; in rubella, 342; in scarlet fever, 321; in status lymphaticus, 547; in syphilis, 366; in tuberculous affections, 373; in whooping cough, 547.
Lymph nodes, agglutination of, producing tumor masses in neck or abdomen, 547; infection of, in simple adenitis, 542; inflammation of, in diphtheria, 304.
Lymph-node tuberculosis. *See Tuberculosis.*
Lymphocytosis, in lymph-node tuberculosis, 384; in lymphoid form of leukemia, 530; in acute poliomyelitis, 649; in simple anemia, 523.
Lymphoid ring, 417, 418.
Lymphosarcoma, diagnosis between, and Hodgkin's disease, 541.
Lymphoid tissue, irritability of, in measles, 342.

M

Malaria, 279.
Malformations of rectum and anus, 233; atresia of anus, 233; congenital absence of the rectum, 233; fissure of the anus, 235; polypus of the rectum, 234; proctitis, 236; prolapse

of the rectum, 234; spasm of the anus, 236.
MARASMUS, 200.
MASSAGE, 53.
MASTOIDITIS, 717.
MEASLES, 332; blood changes in, 336; complications in, 336, 340; contagious period in, 333; convalescent stage in, 336; definition of, 339; diet in, 339; drugs in, 339; enanthem stage in, 334; etiology of, 332; exanthem stage in, 335; immunity from, 333; incubation period in, 333; method of bringing out eruption in, 340; microorganism in, 332; mortality from, 338; nervous symptoms in, 336; pathology of, 338; prognosis of, 338; prophylaxis in, 338; quarantine in, 339; sequelæ of, 337; susceptibility to, 332; symptomatology of, 334; temperature curve in, 335; treatment of, 339; urine findings of, 336.
MEAT PREPARATIONS, 129.
MEDICAL TREATMENT IN INFANCY AND CHILDHOOD, 38.
MENINGITIS, 626.
MENINGITIS, CEREBRO-SPINAL. *See Meningitis, meningococcus.*
MENINGITIS, MENINGOCOCCUS, 631; blood picture in, 634; "carriers" of, 631; complications of, 635; course of, 635; diagnosis of, 634; duration of, 635; etiology of, 631; fever in, 636; microorganism in, 631; mortality from, 635; pathology of, 631; prognosis in, 635; prophylaxis in, 636; sequelæ of, 636; serum treatment of, 636; symptomatology of, 632; symptomatic treatment of, 638; treatment of, 636.
MENINGITIS, PURULENT, 638; definition of 638; diagnosis of, 639; influenza form of, 640; microorganisms present in, 639; mortality from, 639; pneumococcus form of, 639; staphylococcus form of, 639; streptococcus form of, 639; symptomatology of, 639; treatment of, 640; typhoid form of, 640.
MENINGITIS, TUBERCULOUS, 626; diagnosis of, 629; fever in, 628; microorganisms in, 630; mortality from, 627; pathology of, 626; prognosis of, 630; symptomatology of, 627; treatment of, 630.
MENINGOCELE, 621.
MENSTRUAL IRREGULARITIES IN TUBERCULOSIS, 383.
MENTAL PRECOCITY, association of, with physical development, 7, 8, 9.
MERCURY, inunction of, in syphilis, 370.
MICROBLASTS, 516.
MICROCEPHALIC IDIOCY, 625; treatment of, 626.
MICROCYTES, 515; in chlorosis, 527; in pseudoleukemia, 525.
MIGRAINE, 258.
MILK, CONDENSED, 125; cause of rickets, 237; cause of scurvy, 246; value of, as artificial food, 126.
MILK, BREAST. *See Milk, human.*
MILK, COW'S, 118; carbohydrates in, 135, 137; casein in, 135; "certified," 119; cleanliness in care of, 118; composition of, 137; fat in, 135, 137; food value of, 100; home modification of, 135; inorganic constituents of, 104; inspection of, 118; laboratory modification of, 141; malted, 128; pasteurization of, 120; pathogenic organisms transmitted by, 177; peptonization of, 121; poisoning by, in intestinal indigestion, 176; use of, with premature infants, 68; raw, a cause of scurvy, 247; Rotch method of modifying, 141; skimmed, 124; sterilization of, 119; sugar in, 135; tuberculosis transmitted by, 376; typhoid fever transmitted by, 268, 275.
MILK DIET, effect of, on orthostatic albuminuria, 570; in measles, 339; in acute nephritis, 580; in scarlet fever, 329; in typhoid fever, 276.
MILK, HUMAN, advantages of, in enteric infection, 193, 195; advantages of, in summer heat, 181; alexins in, 107; caloric value of, 102; carbohydrates in, 103; casein in, 102; composition of, 107; composition of colostrum in, 108; digestive ferments in, 106; eczema associated with, 722; effect of nursing upon, 113; fat in, 100; food value of, 101; gastrointestinal indigestion associated with, 175; importance of, in bronchopneumonia, 465; importance of, in infantile atrophy, 200; importance of, to premature infants, 67; importance of, in pseudoleukemia, 526; importance of, in syphilis, 309; inorganic constituents of, 104; modification of, 111; proteins in, 102; relation of, to human feeding, 100; sugar in, 103; water in, 105; wholesomeness of, determined, 105.
MILK, MALTED, 128; a cause of rickets, 237, 243; in diet of typhoid fever, 274.
MILK OF MAGNESIA, therapeutic use of,

in eczema, 722; in acute nephritis, 579; in pseudoleukemia, 526.
MILK, MODIFIED, 135; value of, in syphilis, 369.
MILK, PASTEURIZED, 120; scurvy due to, 246.
MILK, PEPTONIZED, 121; value of, in pseudoleukemia, 526.
MILK, STERILIZED, 119; scurvy due to, 246.
MILK SUGAR, intestinal indigestion due to, 103, 125; proportion of, in cow's milk, 103; proportion of, in human milk, 103; value of, in artificial feeding, 125.
MILIARY TUBERCULOSIS, 387; treatment of, 401.
MILLS, on movements of the eyes in infants, 690.
MINERAL SALTS, necessity of, to nutrition, 105; presence of, in milk, 104.
MINOR, on the geographical distribution of scarlet fever, 316.
MITRAL REGURGITATION, 499; association of, with mitral stenosis, 501; prognosis of, 502; treatment of, 503.
MITRAL STENOSIS, 500; association of, with mitral regurgitation, 501; prognosis of, 502; treatment of, 503.
MIXED FEEDING, 114; use of, in hot weather, 181.
MONGOLIAN IDIOCY, 624; diagnosis between, and idiocy of cretinism, 554; treatment of, 625.
MONOPLEGIA, 614.
MONSEL'S SOLUTION, 540.
MONTI, on temperature of milk in infant feeding, 120.
MORO TUBERCULIN TEST, diagnostic value of, 36; in lymph-node tuberculosis, 385; in tuberculous meningitis, 627, 629; technique of, 36.
MORPHIN, therapeutic use of, in chorea, 701; in acute cardiac dilatation, 498; in eclampsia, 683; in acute endocarditis, 493; in enteric infection, 195; in Henoch's purpura, 587; in intestinal hemorrhage in typhoid fever, 278; in intestinal indigestion, 188; in kidney tumor, 589; in meningococcic meningitis, 638; in acute nephritis, 580, 581; in acute pericarditis, 513; in purulent meningitis, 640; in recurrent vomiting, 256; in variola, 348; in whooping cough, 294.
MORSE, on gastrointestinal indigestion as a cause of toxic nephritis, 572; on indirect contagion in whooping-cough, 287; on temperature curve in typhoid fever, 270; on toxic nephritis in pneumonia and meningitis, 572.
MORTALITY, in appendicitis, 227; in bronchopneumonia, 464; causes of, in infancy, 1; in congenital dilatation of the colon, 209; in congenital hypertrophy of the pylorus, 169; in cystopyelitis, 586; in diphtheria, 301; in diplegia, 615; in Hodgkin's disease, 542; in hydrocephalus, 618, 620; in kidney tumor, 589; in lobar pneumonia, 449; in measles, 388; in meningococcic meningitis, 635; in paraplegia, 615; in acute anterior poliomyelitis, 650; among premature infants, 65; reduction of, in diphtheria, under antitoxin treatment, 302; reduction of, in meningitis under Flexner serum, 637; reduction of, in small-pox under vaccination, 350; in scarlet fever, 325; in septic endocarditis, 491; in septic infection of newborn, 79; in status lymphaticus, 548; in stomatitis gangrenosa, 153; in tetanus neonatorum, 91; in tuberculous meningitis, 627, 630; in typhoid fever, 275; in ulcerative endocarditis, 494; in variola, 350; in whooping cough, 291.
MOVEMENTS, PASSIVE AND ARRESTED, therapeutic uses of, 53.
MUCOUS PATCHES IN THE NEW-BORN, 363, 364.
MUCUS, SWALLOWING OF, a cause of intestinal disorders, 177.
MUMPS, 357; complications of, 359; contagious period in, 358; definition of, 357; diagnosis of, 359; diet in, 360; drugs used in, 360; etiology of, 357; microorganisms in, 357; orchitis in, 360; prophylaxis in, 360; sources of infection in, 357; symptomatology of, 358; temperature curve in, 359; transmission of, 357; treatment of, 360.
MURMURS, CARDIAC, in aortic regurgitation, 501; in aortic stenosis, 501; in acute cardiac dilatation, 496; in definite interventricular septum, 487; in diagnosis between endocarditis and pericarditis, 490; in acute endocarditis, 490; functional, 507; in mitral regurgitation, 499; in mitral stenosis, 561; in myocarditis, 495; in chronic pericarditis, 514; in persistent patulous ductus arteriosus, 488; in scarlet fever, 325; systolic, 486; in stenosis of the pulmonary artery, 487.

Murmurs, friction, in acute pericarditis, 510; in chronic pericarditis, 514; in pleurisy, 475.
Murmurs, hemic, in anemia, 523; in chorea, 697, 700; in functional heart disease, 508; in habit spasm, 702.
Murphy, on aspiration for empyema, 480.
Muscular contractures, in chorea, 698; in habit spasm, 702; in infantile cerebral palsies, 613, 615; in acute anterior poliomyelitis, 646; in tetany, 686.
Muscular exercise, therapeutic value of, 52.
Muscular system, development of, 20.
Musser, on angina sine dolor, 497.
Mustard plaster, therapeutic use of, in lobar pneumonia, 451; in pleurisy, 478; objections to, in broncho-pneumonia, 468.
Myelocytes, 517; in leukemia, 530; in pernicious anemia, 529.
Myelitis, 653; definition of, 653; disseminated forms of, 656; etiology of, 653; microorganisms in, 653; paralysis in, 654, 655; prognosis of, 655; serum treatment of, 656; symptomatology of, 653; syphilitic, 655, 657; temperature curve in, 656; transverse forms of, 653; tuberculous form of, 655, 656.
Myocarditis, 494; as cause of acute cardiac dilatation, 496; as complication of bronchopneumonia, 463; as complication of acute articular rheumatism, 404; etiology of, 494; interstitial form of, 495; parenchymatous form of, 495; prognosis of, 495; prophylaxis of, 497; symptomatology of, 495; treatment of, 498.
Myxedema. *See Endemic cretinism.*

N

Nasal douche, 47.
Nasal obstructions, pavor nocturnus due to, 672, 675.
Nasal mucous membrane, diseases of, 411.
Nasal sinuses, disease of, 448.
Nauheim baths, in treatment of valvular heart disease, 505.
Neisser's gonococcus. *See Gonococcus of Neisser.*
Neoplasms of larynx, 435.
Nephritis, acute, 571; albuminuria in, 576; casts in, 576; complications in, 577; as complication of diphtheria, 300; as complication of purpura, 534, 535; as complication of scarlet fever, 324; course of, 577; definition of, 570; diet in, 580; dropsy in, 576; drugs used in treatment of, 581; idiopathic form of, 576; microorganisms present in, 571; mycotic form of, 574; prophylaxis, 578; sources of infection in, 572; symptomatology of, 575; temperature curve in, 577; toxic form of, 572; treatment of, 578; uremia in, 576; urine findings in, 576; varieties of, 572.
Nephritis, chronic diffuse, 582; treatment of, 582.
Nephritis, mycotic, 574.
Nephritis, toxic, 572; treatment of, 573.
Nerves, diseases of peripheral, 661.
Nervous diseases, general, 669.
Nervous disturbances, in adenoid disease, 425; in anemia, 523; in chlorosis, 527; in congenital idiocy, 623; in enteric infection, 192; eruption of permanent teeth accompanied by, 146; in influenza, 310; in acute intestinal indigestion, 184; mother's milk affected by, 111; in acute nephritis, 581; in purpura, 532; in rickets, 240; in scarlet fever, 325; in septic infection, of new-born, 78; due to thyroid intoxication, 557, 558; in typhoid fever, 272; due to uric acid, 562.
Nervous system, development of, 23; excessive activity injurious to, 6; imperfect development of, in premature infants, 62; shock to, dangerous in status lymphaticus, 550.
Nestlé's food, 127; advantages of, in summer, 181; use of, in acute gastritis, 162; use of, in chronic gastritis, 173; use of, in intestinal indigestion, 178; rickets caused by, 237, 243; use of, in typhoid fever, 277.
Netter, on pneumococcus, as cause of pleurisy in child, 470.
Neuritis, multiple, 661; definition of, 661; diagnosis of, 663; diet in, 663; drugs used in treatment of, 663; etiology of, 661; pathology of, 661; symptomatology of, 662; treatment of, 663.
Neuritis, optic, as symptom of brain abscess, 618; as symptom of brain tumor, 617.
Neurotonic reaction, 688.
Neutrophiles, 517.
New-born infant, albuminuria in, 568;

asphyxia in, 69; bronchopneumonia in, 464; care of, 2; cephalhematoma in, 97; clothing of, 5; congenital atelectasis in, 75; congenital hernia in, 86; congenital syphilis in, 83, 361; dermatitis exfoliativa in, 79; diseases of, 69; eclampsia in, 680, 681; epidemic hemoglobinuria in, 82, 564; epistaxis in, 415; erysipelas in, 80; eyes in, 3; facial paralysis in, 98; acute fatty degeneration in, 83; fresh air necessary for, 4; gonococcus vulvovaginitis in, 591; heat-regulating apparatus in, 25; hematoma of sterno-cleido-mastoid muscle in, 98; hematuria in, 563; hemorrhage in, 81; icterus in, 92; inanition fever in, 87; incontinence of urine in, 561; mastitis in, 87; melena neonatorum in, 82; nervous system in, 23; occlusion of bile-ducts in, 93; ophthalmia in, 94; peculiarities of respiratory apparatus in, 410; rest for, 4; sepsis in, 263; sleep for, 671; special senses in, 22; septic infections in, 75; structure of spine in, 19; suppression of urine in, 561; temperature in, 25; tetanus in, 89; typhoid fever in, 269; umbilical affections in, 84; umbilical hernia in, 85; weight of, 15.

NIGHT-TERROR, 672; symptomatology of, 674; treatment of, 675.

NIPPLES, CARE OF, 113; in stomatitis mycosa, 149.

NITRE PAPER inhalation of fumes from, in asthma, 712.

NITROGLYCERIN, therapeutic use of, in asthma, 712; in acute cardiac dilatation, 498; in lobar pneumonia, 463.

NOMA. *See Stomatitis gangrenosa.*

NORMOBLASTS, 515.

NORTHRUP, experiments by, showing danger of food pneumonia, 306; on fresh air in bronchopneumonia, 465; on fresh air in lobar pneumonia, 450; on fresh air as therapeutic measure, 43, 44; on rest cure in neurotic disturbances, 53; on scurvy in infants, 246; on sleeplessness in infants, 4.

NOSE, disease in accessory sinuses of, as complication of lobar pneumonia, 448; disinfection of, a protection against recurring attacks of endocarditis, 493; foreign bodies in, 416; removal of diseased tissue from, a protection against tuberculosis, 395; ulceration of, in late hereditary syphilis, 367.

NURSERY, 5.

NURSING OF INFANTS, adenoid growths an obstacle to, 426; retropharyngeal abscess, an obstacle to, 428; rules for, 113.

NUX VOMICA, TINCTURE OF, therapeutic use of, in asthma, 713; in acute endocarditis, 494.

NYSTAGMUS AND HEAD-NODDING, 689; character of movements in, 689; definition of, 689; etiology in, 690; prognosis in, 691; treatment of, 691.

O

OBSCURE FEVER, 263; use of antipyretics in, 266; diet in, 266; inanition fever a cause of, 263; intestinal toxemia a cause of, 265; lobar pneumonia a cause of, 264; otitis media a cause of, 264; pyelocystitis a cause of, 264; sepsis a cause of, 263; septic infection a cause of, 265; treatment of, 265; tuberculosis a cause of, 264; typhoid fever a cause of, 265.

O'DWYER, DR. JOSEPH, on intubation for relief of laryngeal diphtheria, 305.

O'DWYER INTUBATION SET, 305.

OILSKIN JACKET, use of, in bronchopneumonia, 468; use of, in acute catarrhal bronchitis, 442; contraindications to, in lobar pneumonia, 451.

OINTMENTS IN TREATMENT OF ECZEMA, 724.

OLIGOCHROMEMIA, 516.

OLIGOCYTHEMIA, 516.

OPHTHALMIA, diphtheritic, 299.

OPHTHALMIA NEONATORUM, 94; diagnosis of, 95; etiology of, 94; prognosis of, 96; prophylaxis in, 96; symptomatology of, 95; treatment of, 96.

OPISTHOTONOS, in lobar pneumonia, 449; in meningococcic meningitis, 632; in tuberculous meningitis, 628.

OPIUM, therapeutic use of, in appendicitis, 227; in brain abscess, 618; in acute endocarditis, 493; in enteric infection, 195; during infancy, 40; in influenza, 314; in acute intestinal indigestion, 188; in kidney tumor, 589; in lobar pneumonia, 453; in meningococcic meningitis, 638; in peritonitis, 232; in acute anterior poliomyelitis, 651; in recurrent vomiting, 256; in typhoid fever, 278; in whooping-cough, 294.

OPIUM, contraindications to use of, in bronchopneumonia, 467; in cough of tuberculosis, 400; in measles, 340; in multiple neuritis, 664.

"OPSONINS," 55.

Opsonic index, 56.
Orange juice, use of, in constipation, 206, 207; in diet of infants, 137, 142, 143; in scurvy, 249; in typhoid fever, 277.
Orchitis, as complication of mumps, 359.
Orthopedics, in treatment of hemiplegia, 615; in treatment of hereditary ataxia, 658.
Orthostatic albuminuria, 568; definition of, 568; diet in, 570; etiology of, 569; prognosis of, 570; symptomatology of, 569; treatment of, 570; urine findings in, 569.
Osler, on appearance of skin in Henoch's purpura, 534; on aspiration in acute pericarditis, 513; on cardiac murmurs in chorea, 700; on cause of pain in Henoch's purpura, 534; on sarcomatous nature of kidney tumors in child, 587; on typhoid fever in autumn, 268.
Osteochondritis, syphilitic, 362.
Osteomyelitis, acute, as complication of bronchopneumonia, 463; as complication of lobar pneumonia, 448; mistaken for articular rheumatism, 405.
Otitis, chronic, in late hereditary syphilis, 367.
Otitis interna, headache from, 709.
Otitis media, 714; due to adenoid disease, 425; brain abscess caused by, 617, 618; bronchopneumonia complicated by, 463; acute catarrhal bronchitis, complicated by, 439; diphtheria complicated by, 300; etiology of, 714; facial paralysis caused by, 664, 665; influenza complicated by, 311; intermittent fever caused by, 715; lobar pneumonia complicated by, 448; mastoiditis caused by, 717; microorganisms present in, 714; mumps complicated by, 359; obscure fever caused by, 264; operative treatment for, 716; otoscopic examination in, 715; prognosis of, 716; prophylaxis of, 716; scarlet fever complicated by, 324, 331; sources of infection in, 714; symptomatology of, 714; temperature curve in, 715; tonsillitis complicated by, 419; treatment of, 716; typhoid fever complicated by, 274.
Out-door play, therapeutic value of, 54.
Ovaritis, as complication of mumps, 359.
Over-feeding, as a cause of indigestion in infancy, 133, 175.
Oxybutyric acid, presence of, in urine, 565.
Oxalic acid, excess of, in urine in orthostatic albuminuria, 569.
Oxygen, inhalation of, in asphyxia neonatorum, 73, 74; in bronchopneumonia, 466; in lobar pneumonia, 453; in tuberculous bronchopneumonia, 466.
Oxyuris vermicularis, 217; diagnosis of, 218; treatment of, 218.

## P

Packs, cold, use of, in lobar pneumonia, 451; objections to, in acute catarrhal bronchitis, 442; use of, in obscure fevers of childhood, 267; restricted use of, in bronchopneumonia, 468; use of, in scarlet fever, 329; temperature of, 45; therapeutic value of, 45; use of, in typhoid fever, 277.
Packs, hot, use of, in acute nephritis, 579.
Packard, Dr. Frederick, on tonsillitis preceding endocarditis, 490; on tonsillitis preceding infectious diseases, 417.
Padded basket, use of, for premature infants, 66.
Palate, destructive ulceration of, in late hereditary syphilis, 367.
Palpation, value of, in physical examination, 30.
Paracentesis, in ascites due to valvular heart disease, 506; in otitis media, 716.
Paralysis, cardiac, in diphtheria, 299; in multiple neuritis, 663.
Paralysis, facial, 664; diagnosis of, 665; drugs in, 666; etiology of, 664; in new-born, 97; operative treatment for, 666; in acute anterior poliomyelitis, 645; prognosis of, 665; symptomatology of, 664; treatment of, 666.
Paralysis, motor, in hysteria, 705; in multiple neuritis, 662; in myelitis, 654; in acute anterior poliomyelitis, 647; in spina bifida, 660.
Paralysis, post-diphtheritic, 299, 307.
Paralysis, pseudo, in syphilis, 365.
Paralysis, splanchnic, in diphtheria, 299, 307; in influenza, 312, 314; in lobar pneumonia, 448.
Paraphimosis, 596.
Paraplegia, 614; treatment of, 615.
Parasites, intestinal, 211; ascaris lumbricoides, 215; bothriocephalus

latus, 212; hymenolepis nana, 212; oxyuris vermicularis, 217; tenia elliptica, 213; tenia saginata, 211; tenia solium, 212; trichuris trichiura, 217.

PARATHYROID METABOLISM, defect in, a cause of muscle spasm, 686.

PARATYPHOID FEVER, diagnosis between, and typhoid, 275.

PAREGORIC, therapeutic use of, for cough in measles, 339; in earache, 716; in enteric infection, 195; in acute gastritis, 162; in acute intestinal indigestion, 188; in kidney tumor, 689; in lobar pneumonia, 463; in pleurisy, 478; in tuberculous peritonitis, 410; in typhoid fever, 278; in whooping-cough, 294.

PARK, DR. W. H., on diphtheria in animals, 295; on management of tenement houses, 182.

PARKE, DR. THOMAS D., on recurrent vomiting, 255.

PAROTITIS, EPIDEMIC. *See Mumps.*

PAROTITIS, as complication of typhoid fever, 274, 276.

PASTEURIZED MILK, 120.

PATELLAR REFLEX, examination of, in illness, 31; in diphtheria, 299.

PAVOR NOCTURNUS, 671; central type of, 672; clinical pictures of, 674; definition of, 671; etiology of, 671; exciting causes of, 672; predisposing causes of, 671; prognosis of, 675; symptomatic type of, 674; symptomatology of, 672; treatment of, 675.

PAVY, on orthostatic albuminuria, 568.

PEACOCK AND KEITH, on stenosis of pulmonary artery, 487.

PEDICULOSIS CAPILLITII, 736; treatment of, 737.

PEMPHIGOID ULCERATIONS, as complication of measles, 338.

PEMPHIGUS NEONATORUM, 732; diagnosis between, and syphilitic pemphigus in new-born, 364; treatment of, 733.

PEMPHIGUS, SYPHILITIC, 364.

PEPSIN, elixir of, as vehicle for drugs, 39; essence of, as vehicle for drugs, 39.

PERCENTAGE FEEDING, value of, 129.

PERCENTAGES IN FOOD FORMULAS, 139.

PERCUSSION, value of, in physical examination of child, 32.

PERICARDITIS, ACUTE, 509; complicating acute articular rheumatism, 404; complicating bronchopneumonia, 463; course of, 512; definition of, 509; diagnosis of, 511; diet in, 512; etiology of, 509; complicating Henoch's purpura, 534; complicating lobar pneumonia, 448; microorganisms present in, 509; operative treatment for, 513; pathology of, 509; physical signs of, 510; prognosis of, 512; prophylaxis of, 512; sources of infection in, 509; temperature curve in, 510; treatment of, 512; varieties of, 509.

PERICARDITIS, CHRONIC, 513; diagnosis of, 514; etiology of, 514; physical signs in, 514; results of, 514; symptomatology of, 514; treatment of, 514.

PERIESOPHAGEAL ABSCESS, 157.

PERONEAL TYPE OF PROGRESSIVE MUSCULAR DYSTROPHY, 668.

PERINEPHRITIS, 590.

PERIOSTITIS, due to late hereditary syphilis, 367.

PERITONITIS, ACUTE, 228; complicating appendicitis, 229; etiology of, 228; exciting causes of, 228; complicating lobar pneumonia, 448; microorganisms present in, 228; pathology of, 229; physical examination in, 231; prognosis of, 231; symptomatology in, 229; temperature curve in, 230; treatment of, 231.

PERITONITIS, TUBERCULOUS, diagnosis of, 390; pathology of, 381; treatment of, 401.

PERITONSILLAR ABSCESS, 421.

PERLÈCHE, 154; treatment of, 155.

PERMANGANATE OF POTASH. *See Potassium permanganate.*

PERNICIOUS ANEMIA, 528.

PEROXID OF HYDROGEN, therapeutic use of, in acute anterior poliomyelitis, 651; in tonsillitis, 423; in umbilical infection of the new-born, 80.

PERSISTENT PATULOUS DUCTUS ARTERIOSUS, 488.

PERTUSSIS. *See Whooping-Cough.*

PETERSON, on rotary head movements in infants, 689.

PETIT MAL, 692.

PFAUNDLER, on agglutination reaction in cystopyelitis, 584.

PFEIFFER, discovery by, of influenza bacillus, 308, 309.

PFEIFFER'S BACILLUS. *See Influenza bacillus.*

PHARYNGITIS, in adenoid disease, 425; in influenza, 310; preceding acute laryngitis, 430; in acute rhinitis, 413.

PHARYNX, destructive ulceration of, in late hereditary syphilis, 367; diseases of, 424; removal of diseased tissue

from, a protection against tuberculosis, 395.

PHENACETIN, therapeutic use of, contraindicated in bronchopneumonia, 467; in acute catarrhal bronchitis, 441; in fevers of infancy and childhood, 267; in headache, 710; in influenza, 313; in kidney tumor, 589; in lobar pneumonia, 452; in malaria, 286; in measles, 340; in mumps, 340; in multiple neuritis, 664; in acute anterior poliomyelitis, 651; in rubella, 343; in tonsillitis, 278; in tuberculosis, 400; contraindicated in typhoid fever, 278; in varicella, 357; in urticaria, 727.

PHIMOSIS, 596; association of, with enuresis, 599, 601; association of, with pseudomasturbation, 608.

PHOSPHATE OF SODA. *See Sodium phosphate.*

PHOSPHORUS, presence of, in milk, 104; therapeutic use of, in rickets, 245; toxic nephritis due to, 573, 575; uses of, to body, 105.

PHYSICAL EXAMINATION, of sick child, 29.

PHYSICAL SIGNS, in aortic regurgitation, 501; in aortic stenosis, 501; in appendicitis, 224; in acute bronchitis, 240; in bronchopneumonia, 460; in acute cardiac dilatation, 496; in failure of compensation, 502; in intestinal intussusception, 221; in lobar pneumonia, 458, 460; in lymph-node tuberculosis, 384; in mitral regurgitation, 499; in mitral stenosis, 501; in myocarditis, 495; in acute pericarditis, 510; in chronic pericarditis, 514; in peritonitis, 231; in pleurisy with effusion, 472; in splenic enlargement, 551; in sporadic cretinism, 553; in thyroid enlargement, 547; in tuberculous bronchopneumonia, 388, 462.

PHYSIOLOGICAL ALBUMINURIA, 567.

PHYSIOLOGICAL GASTRIC INCOMPETENCY, 158.

PICA, 704; treatment of, 705.

PICK'S DISEASE, 514.

"PINK EYE," 413.

PLASMODIUM MALARIÆ, 279.

PLAYGROUNDS, hygienic value of, 10.

PLEURISY, 470; aspiration for, 479; complicating bronchopneumonia, 462; definition of, 470; diet in, 478; displacement of organs in, 475, 476; drugs used in treatment of, 478, 479; with effusion, 471, 473; etiology of, 470; exploratory puncture in, 476; fluoroscopic examination in, 476; forms of, 471; complicating lobar pneumonia, 448; microorganisms present in, 470; pain in, 473; pathology of, 471; physical signs in, 475; respiration in, 473; sources of infection in, 470; surgical treatment of, 479; symptomatology of, 472; temperature curve in, 472; treatment of, 472; treatment of, during convalescence, 482; X-ray picture in, 476.

PNEUMOCOCCUS PERITONITIS, 228, 231, 232.

PNEUMOCOCCUS VACCINE, therapeutic use of, 58.

PNEUMONIA, BRONCHO-. *See Bronchopneumonia.*

PNEUMONIA, DEGLUTITION, 462.

PNEUMONIA, FOOD, risk of, after intubation in laryngeal diphtheria, 306.

PNEUMONIA, LOBAR, 442; antipyretics in, 451; auscultation in, 448; blood changes in, 447; complications in, 448; cough in, 448; crisis in, 445; danger from, in infancy, 6; diet in, 450; differential diagnosis in, 449; drugs in, 452; etiology of, 442; hygienic treatment of, 450; local applications in, 451; complicating measles, 337; medical treatment of, 452; microorganisms present in, 442; mortality from, 2, 449; obscure fevers in infancy due to, 264; pathology of, 443; percussion in, 448; physical signs in, 447; prognosis of, 449; respiration in, 446; sources of infection in, 443; stimulants in, 451; symptomatology of, 444; temperature in, 261, 444, 445; treatment of, 449; treatment of, during convalescence, 453; complicating typhoid fever, 274; urine findings in, 447; complicating whooping-cough, 294.

POIKILOCYTOSIS, 516.

POISONS, IRRITANT, cause of enterocolitis, 189; cause of hemoglobinuria, 564.

POLIOENCEPHALITIS. *See Acute anterior poliomyelitis.*

POLIOMYELITIS, ACUTE ANTERIOR, 641; abortive type, 645; ataxic type, 646; bulbar type, 645; chronic changes in, 644; definition of, 641; descending type of, 645; diagnosis of, 649; electrical reactions in, 649; encephalitic type, 646; epidemic form of, 642; etiology of, 641; immunity from, 644; meningeal type, 646; mortality from, 650; muscular atrophy in, 648; paral-

ysis in, 647; pathology of, 643; polyneuritic type, 647; pontine type, 645; prophylaxis of, 651; prognosis of, 650; sources of infection in, 642; spinal poliomyelitic type, 646; sporadic form of, 642; symptomatology of, 644; transmission of, 641; treatment of, 651; treatment of paralysis in, 652; use of urotropin in, 651.

POLYCHROMASIA, 516.

POLYCYTHEMIA, 516.

POLYPNEA in young children, 27.

POLYPUS OF THE RECTUM, 234.

PORTER, W. T., on comparative rate of growth in children at public schools, 16; on physical basis of precocity and dullness, 7.

PORTER AND FLEISCHNER, report of recurrent vomiting associated with cystopyelitis, 585.

POSITION, significance of, in examination of sick child, 30.

POSTURAL ALBUMINURIA. *See Orthostatic albuminuria.*

POSTNATAL PALSIES, 611.

POTASSIUM ACETATE, therapeutic use of, in pleurisy with effusion, 478.

POTASSIUM BICARBONATE, therapeutic use of, in enuresis, 602.

POTASSIUM CARBONATE, therapeutic use of, in urethritis in male child, 596.

POTASSIUM CHLORATE, hemoglobinuria caused by, 564; hematuria caused by, 563; purpura caused by, 532; therapeutic use of, in stomatitis ulcerosa, 151; toxic nephritis caused by, 573, 575.

POTASSIUM CITRATE, therapeutic use of, in cystopyelitis, 586.

POTASSIUM IODID, therapeutic use of, in brain tumors, 617; in hydrocephalus, 620.

POTASSIUM PERMANGANATE, therapeutic use of, in gonococcus vulvovaginitis, 594.

POTT'S DISEASE, 391; examination for, 31; myelitis as complication of, 653, 654, 655, 656.

POVERTY, tuberculosis a cause of, 375.

POYNTON, on presystolic murmur in mitral regurgitation, 500.

PREMATURE INFANTS, 62; artificial feeding for, 68; breast feeding for, 67; digestive system in, 64; feeding of, 67; heat-regulating mechanism, 25, 62; hereditary disease in, 64; incubator for, 66; nervous system in, 62; padded basket for, 66; physical peculiarities of, 62; prognosis for, 65; quantity of food for, 68; susceptibility to reinfection in, 64; treatment of, 65; weight of, 65; wet nurse for, 68.

PRENATAL PALSIES, 610.

PREPUCE, ADHERENT, 596; enuresis associated with, 599, 601; pseudomasturbation associated with, 608.

PRESSURE SIGNS IN LYMPHNODE TUBERCULOSIS, 385.

PRIESSNITZ'S APPLICATION, in treatment of bronchopneumonia, 468; in treatment of lobar pneumonia, 451.

PROCTITIS, 236.

PROLAPSE OF RECTUM, 234.

PROGRESSIVE MUSCULAR DYSTROPHY, 666; definition of, 666; Erb's juvenile type of, 668; etiology of, 666; Landouzy-Déjerine type of, 668; peroneal type of, 668; pseudohypertrophic type of, 666; symptomatology of, 666; treatment of, 669.

PROPRIETARY FOODS, 127; in causation of rickets, 237; in causation of scurvy, 246; value of, in artificial feeding, 127; value of, in chronic gastritis, 172; value of, in hot weather, 182; value of, in typhoid fever, 277.

PROTEIN, digestibility of, 135; indigestion from, 186; nutritional value of, 103; proportion of, in beef juice, 129; proportion of, in broth, 129; proportion of, in buttermilk, 122, 123; proportion of, in Finkelstein's albumin milk, 123; proportion of, in malt soups, 124; proportion of, in milk, 101; proportion of, in skimmed milk, 124.

PROTEUS VULGARIS, in causation of enteric infection, 178.

PRYOR, on development of bony framework in child, 19.

PSEUDO-DIPHTHERITIC BACILLUS, in causation of simple vulvovaginitis, 595.

PSEUDO-HYPERTROPHIC FORM OF PROGRESSIVE MUSCULAR DYSTROPHY, 666.

PSEUDOLEUKEMIA OF INFANTS, 524; age as predisposing factor in, 524; blood changes in, 525; definition of, 524; diet in, 526; differential diagnosis in, 525; drugs used in treatment of, 526; etiology of, 524; prognosis of, 526; symptomatology of, 525; temperature curve in, 525; treatment of, 526.

PSEUDOMASTURBATION, 603; age as etiological factor in, 604; definition of, 603; direct causes of, 607; drugs used in treatment of, 609; environment as

etiological factor, 606; epilepsy not related to, 607; etiology of, 604; general treatment of, 609; habit, as etiological factor in, 606; mechanical treatment of, 609; neurotic inheritance an etiological factor in, 606; prognosis of, 607; sex as an etiological factor in, 606; symptomatology of, 603; treatment of, 607.

PSYCHIC EPILEPSY, 693.

PSYCHOTHERAPY, value of, in nervous disorders, 54; in whooping-cough, 292.

PUBERTY, rapid growth in height preceding, 17; rapid growth in weight preceding, 16; reflex disturbances at, 13; thyroid gland excessively active at, 17.

PUBLIC SCHOOL SYSTEM, hygienic defects in, 8.

PULMONARY TUBERCULOSIS. *See Tuberculosis of the lungs under Tuberculosis.*

PULSE RATE, instability of, in infancy, 484; peculiarities of, in myocarditis, 495; peculiarities of, in typhoid fever, 270; rapidity of, in bronchopneumonia, 457; rapidity of, in pleurisy, with displacement of the heart from effusion, 473; ratio between, and temperature in lobar pneumonia, 444.

PUNCTURE, EXPLORATORY, in pleurisy with effusion, 476.

PUNCTURE, LUMBAR. *See Lumbar.*

PURPURA, 531; character of eruption in, 531; definition of, 531; diet in, 535; drugs in causation of, 532; drugs used in treatment of, 536; etiology of, 532; fulminans, 523, 537; hemorrhagica, 533, 536; Henoch's purpura, 534, 537; hydrotherapy for, 536; medical treatment of, 536; rheumatica, 535, 537; simplex, 532; symptomatica, 532; temperature curve in p. hemorrhagica, 533; treatment of, 535; treatment of special forms, 536; varieties of, 532.

"PURPURAS," 531.

PURULENT MENINGITIS. *See Meningitis, purulent.*

PUTNAM, on arsenic as cause of neuritis in children, 661; on night terror, 673, 675.

PYELITIS, diagnosis between, and malaria, 284.

PYEMIA, diagnosis between, and malaria, 284.

PYELOCYSTITIS, as cause of obscure fever in infancy, 264.

PYRAMIDON, therapeutic use of, in fever of tuberculosis, 400.

Q

QUARANTINE, hygienic reasons for, 6; length of, in diphtheria, 302; in influenza, 312; in measles, 339; in mumps, 358; in acute anterior poliomyelitis, 651; in rubella, 343; in scarlet fever, 327; in varicella, 357; in variola, 348; in whooping-cough, 288.

QUASSIA, use of, for removal of oxyuris vermicularis, 218.

QUININ, therapeutic use of, in asthma, 713; in chlorosis, 528; in chorea, 701; in headache, 710; in influenza, 313; in lobar pneumonia, 452; in malaria, 284; in rhinitis, 415; in tonsillitis, 422; in whooping-cough, 293; purpura caused by, 532.

QUINSY. *See Peritonsillar abscess.*

R

RACHITIS. *See Rickets.*

RADIAL FISSURES, 364, 365.

RADIOGRAPHY. *See X-Ray treatment.*

RANSOHOFF'S METHOD for after-treatment of empyema, 48.

"RASPBERRY EXCRESCENCE," 353.

REACTIONS, ELECTRICAL, in facial paralysis, 665; in multiple neuritis, 663; in acute anterior poliomyelitis, 649; in progressive muscular dystrophy, 667.

RECTAL ENEMATA, therapeutic value of, 51.

RECTAL FEEDING, therapeutic value of, 51.

RECTAL IRRIGATION, therapeutic value of, 46.

RECTAL SUPPOSITORIES, therapeutic value of, 52; in constipation, 207; in scarlet fever, 331; in typhoid fever, 278; objections to, in artificially fed infant, 206.

RECTAL TEMPERATURE, importance of taking, in examination of sick child, 29.

RECTUM, MALFORMATION OF. *See Malformation.*

RECTUM, POLYPUS OF, 234.

RECTUM, PROLAPSE OF, 234; in enteric infection, 191; caused by whooping-cough, 289.

RECURRENT CORYZA, 258.

RECURRENT VOMITING, 251; blood changes in, 254; climate in treatment

of, 257; clinical types of, 254; in cystopyelitis, 285; definition of, 251; diagnosis of, 254; diet in, 254; drugs used in treatment of, 255, 256; etiology of, 251; exciting causes of, 251; hygiene in treatment of, 257; pain in, 253; predisposing causes of, 252; prognosis of, 254; symptomatology of, 252; synonyms for, 251; temperature changes in, 253; treatment of attack of, 255; treatment of interval in, 256; urine findings in, 254.

Red light treatment of small-pox, 349.

Reflex centers, imperfect development of, in premature infants, 64.

Reflex irritation, development of genital organs a cause of, 13; eclampsia due to, 668; eczema due to, 719, 722; enuresis due to, 601; epilepsy due to, 692, 695; headache due to, 709; habit spasm due to, 702; hysteria due to, 705; insomnia due to, 676; laryngismus stridulus due to, 684; masturbation due to, 606, 608; nervous strain due to, 10, 11, 13; night-terror due to, 772; tetany due to, 687.

Reflexes, examination of, in sick child, 31; in acute anterior poliomyelitis, 644; in progressive muscular dystrophy, 607.

Renal calculi, formation of, in infants, 562.

Resorcin, therapeutic use of, in eczema, 723.

Respiration, acceleration of, for slight causes in infancy, 110; artificial, 72; peculiarities of, in child, 410.

Respiratory centers, imperfect development of, in premature infants, 63.

Respiratory movements, examination of, in sick child, 30.

Rest, therapeutic value of, 52; in acute articular rheumatism, 405; in chorea, 700; in control of fever in tuberculosis, 400; in eclampsia, 684; in endocarditis, 495; in infancy, 11, 13, 135; in hysteria, 768; in myocarditis, 498; in new-born, 4; in pericarditis, 535; in acute anterior poliomyelitis, 651; in tuberculous peritonitis, 401; in valvular heart disease, 505.

Rest in bed, therapeutic value of, in acute nephritis, 580; in multiple neuritis, 663; contraindicated in orthostatic albuminuria, 570.

Rest cure, indications for, 52.

Retro-pharyngeal abscess, 427; diagnosis of, 428; microorganisms present in, 427; predisposing causes of, 428; prognosis of, 429; sources of infection in, 427; symptomatology of, 428; temperature changes in, 428; treatment of, 429.

Reuss, experiments by, on value of dextri-maltose preparations, 125.

Rheumatic nodules, 403.

Rheumatic wry neck, 403.

Rheumatism, acute articular, 402; chorea due to, 404, 697; definition of, 402; diagnosis of, 404; diet in attack of, 405; diet in interval of, 406; drugs used in treatment of, 405; erythema multiforme due to, 729; etiological factor in endocarditis, 489; etiology of, 402; heart disturbances in, 404; heredity in causation of, 402; medical treatment of, 405; microorganisms present in, 402; myelitis as sequel to, 653; prognosis of, 405; salicylates in the treatment of, 405; simple anemia in, 404; sources of infection in, 402; symptomatology of, 403; temperature changes in, 405; tonsillitis in, 404; treatment of attack, 405; treatment of interval, 406; valvular heart disease due to, 498, 503.

Rheumatism, scarlatinal, 324, 331.

Rhinitis, acute, 411; adenoid disease a cause of, 425; definition of, 411; drugs used in treatment of, 415; etiology of, 411; acute laryngitis following, 430; microorganisms present in, 411; prophylaxis in, 414; prognosis of, 413; sources of infection in, 411; symptomatology of, 412; temperature changes in, 412, 413; treatment of, 414.

Rhinitis, chronic, 414; treatment of, 415.

Rickets, 237; blood changes in, 242; bony deformities in, 241; congenital form, 242; course of, 242; curative treatment of, 243; definition of, 237; diagnosis of, 242; drugs used in treatment of, 244; eclampsia caused by, 679; gastrointestinal disorders in, 240; head-sweating in, 240; hernia in, 240; general appearance in, 239; late form, 412; morbid anatomy of, 238; previous symptoms in, 240; pathology of, 238; prevention of deformities in, 245; prognosis in, 242; prophylaxis in, 243; symptomatology of, 239; treatment of, 243; treatment of deformities in, 245; tetany caused by, 686; weakness of ligaments and muscles due to, 240.

RINGWORM OF THE TONGUE. *See Geographical tongue.*
ROGER, on situation of systolic murmur in defective interventricular septum, 487.
ROMBERG AND PASSLER, on splanchnoparalysis as a result of pneumococcus toxins, 448.
ROMBERG'S SIGN, 657.
RÖNTGEN RAYS, diagnostic value of, 38; in bone and muscle development and capacity, 19, 20; in bone and joint tuberculosis, 393; in congenital dilatation of the colon, 209; in cystopyelitis, 586; in demonstration of enlarged thymus, 547; in foreign bodies in larynx, trachea, and bronchi, 437; in infective arthritis, 408; in lymph node tuberculosis, 385; in acute pericarditis, 511; in pleurisy with effusion, 476; in persistent patulous ductus arteriosus, 488; in spina bifida, 659; in status lymphaticus, 547.
RÖNTGEN RAYS, therapeutic value of, in Hodgkin's disease, 542; in leukemia, 531; in status lymphaticus, 548, 549; in tinea tonsurans, 735; in thyroid enlargement, 549; rules for use of, in status lymphaticus, 550; technique for use of, in thyroid enlargement, 549.
ROSE POSITION, in operation for retropharyngeal abscess, 429.
ROSE SPOTS, in diagnosis of typhoid fever, 271.
ROTCH, on increase in height during first month of life, 17; on indications for paracentesis in acute pericarditis, 513; on lack of conformity between chronological age and physical development, 20; on modification of milk, 141; practical system by, for grading work of child, 20; on relative frequency with which tuberculosis attacks different joints, 381; report by, on case of meningococcic meningitis in infant six days old, 631; on Röntgenization of wrist bones as indication of bone and muscle development in child, 19; on structure of spine at birth, 19.
ROTCH LABORATORY METHOD OF MODIFYING MILK, 141.
RÖTHELN. *See Rubella.*
ROUND WORM. *See Ascaris lumbricoides.*
ROUX, work done by, upon antitoxin, 302.
RUBELLA, 341; blood changes in, 343; complications in, 343; definition of, 341; drugs used in treatment of, 343; enanthem of, 342; etiology of, 341; exanthem of, 342; immunity from, 341; incubation period in, 341; lymphatic tissue affected in, 342; prognosis of, 343; symptomatology of, 341; temperature changes in, 341; treatment of, 343; urine findings in, 343.
RURÄH, table by, showing causes of nocturnal enuresis, 600; table by, showing differential diagnosis of acute exanthemata, 344.

S

SABOURAUD, on the parasite causing tinea tonsurans, 733.
SACHS, on aphasia associated with left-sided hemiplegia, 613; on epilepsy, 692, 694; on relation of epilepsy to early spastic palsies, 613; on surgical treatment of epilepsies, 696; table by, showing classification of infantile cerebral palsies, 611.
SALICYLATE OF SODIUM, therapeutic value of, in facial paralysis, 666; in acute follicular tonsillitis, 422.
SALICYLATES, diagnostic value of, in rheumatism, 404; therapeutic value of, in acute endocarditis, complicating rheumatism, 493; in pleurisy, with gout tendencies, 478; in prophylaxis of endocarditis, 493; in rheumatism, 405; in valvular heart disease due to rheumatism, 504.
SALICYLIC ACID OINTMENT, therapeutic use of, 42, 43; in congenital ichthyosis, 731; in eczema, 723; in impetigo contagiosa, 732; in pleurisy, 479; in tinea tonsurans, 734.
SALOL, therapeutic use of, in acute catarrhal bronchitis, 441; in chlorosis, 528; in chorea, 701; in cystopyelitis, 587; in acute endocarditis, 494; in influenza, 314; in pernicious anemia, 529; in pseudoleukemia, 526; in tonsillitis, 422; in typhoid fever, 277; in urethritis in male child, 596; in urticaria, 728.
SALT BATHS, therapeutic value of, 46.
SALVARSAN, therapeutic use of, in syphilis, 372.
SANTONIN, therapeutic use of, for ascaris lumbricoides, 216; for oxyuris vermicularis, 218.
SCABIES, 735; treatment of, 736.
SCARLET FEVER, 315; antistreptococcic serum in, 330; cervical adenitis in, 321; climatic influences upon, 315; complications in, 324; contagious period in, 317; convalescence from, 332; defini-

tion of, 315; desquamation in, 322; diagnosis of, 325; diet in, 329; drugs used in treatment of, 330; eruption in, 321, 325; etiology of, 315; exciting causes of, 316; fulminating type of, 323; geographical distribution of, 316; hydrotherapy in, 329; incubation period in, 318; inunctions in, 330, 331; irregular clinical types of, 323; malignant type of, 323; measles complicated by, 338; microorganism in, 316; mild type of, 323; mortality from, 2, 325; mycotic nephritis due to, 574; pathology of, 317; predisposing causes of, 315; prognosis of, 325; prophylaxis of, 327; quarantine in, 327; recurrence of, 323; relapses in, 323; septic type of, 323; sore throat in, 321; sources of infection in, 316; stimulants in, 329; symptomatology of, 318; temperature changes in, 318; tongue in, 321; transmission of, 316; treatment of, 328; treatment of nose and throat in, 330; urine findings in, 322; vomiting in, 318.

SCARLATINA. *See Scarlet fever.*

SCHÖNLEIN'S DISEASE. *See Purpura rheumatica.*

SCHOOL WORK, nervous strain of, 8; habit spasm due to, 702; hysteria due to, 705; insomnia due to, 676; night-terror due to, 672; weight a test of endurance of, 7, 8.

SCHOTTMUELLER'S DISEASE, 494.

SCHULTZE'S METHOD of artificial respiration, 72.

SCORBUTUS. *See Scurvy.*

SCURVY, INFANTILE. *See Infantile.*

SEBORRHEA, character of, 720; treatment of, 726.

SEIBERT, on humidity affecting mortality from intestinal disorders, 180; on ichthyol ointment in scarlet fever, 330.

SEPTICEMIA, complicating diphtheria, 299, 307; complicating scarlet fever, 323, 330; diagnosis between, and malaria, 284.

SEPTIC ARTHRITIS, as a complication in scarlet fever, 324.

SEPTIC INFECTION. *See Sepsis.*

SEPSIS, diphtheria complicated by, 296; fever in second week of life due to, 263; myelitis following, 655; obscure fevers in childhood due to, 265; premature infants liable to, 164.

SEPTIC INFECTIONS IN NEW-BORN, 75; diagnosis of, 78; etiology of, 75; individual symptoms in, 78; prognosis in, 79; sources of infection in, 76; symptomatology of, 77.

SERUM, ANTIMENINGITIC, 636; effect of, upon prognosis, 635; effect of, upon sequelæ, 635; nature of, 60.

SERUM, ANTISTREPTOCOCCIC, therapeutic use of, in diphtheritic septicemia, 307; in hemophilia, 540; in acute infective myelitis, 657; in measles with septic complications, 340; in scarlet fever, 330; in stomatitis gangrenosa, 153; in ulcerative endocarditis, 494.

SERUM, ANTITOXIN, OF DIPHTHERIA. *See Antitoxin.*

SERUM, STANDARDIZED ANIMAL BLOOD, therapeutic use of, in hemophilia, 540.

SERUM TREATMENT, 59; asthmatic patients unsuited for, 713; in diphtheria, 302, 307; in hemophilia, 540; in acute infective myelitis, 657; in measles with septic complications, 340; in meningococcic meningitis, 635; rashes from, mistaken for scarlet fever, 330; in scarlet fever, 330; in stomatitis gangrenosa, 153; in ulcerative endocarditis, 494.

SHELL-FISH, a source of infection in typhoid fever, 268, 275.

SHIGA BACILLUS, association of, with gastroenteric affections, 178.

SILBERMANN, on types of night-terror, 672, 673, 674.

SIGHT, sense of, in infancy, 22.

SILVER NITRATE, therapeutic use of, in local treatment of scarlet fever, 330; by irrigation, in cystopyelitis, 587; in gonococcus vulvovaginitis, 594.

SILVER, COLLOIDAL, therapeutic use of, 43.

SIMPLE SECONDARY ANEMIA, 522.

SKIMMED MILK, 124.

SKIN REACTIONS FROM TUBERCULIN. *See Tuberculin.*

SKIN, inspection of, in illness, 30; itching of, in status lymphaticus, 547; stimulation of, in acute nephritis, 579.

SLEEP, amount of, necessary in childhood, 671; amount of, necessary to newborn, 4; amount of, necessary to school child, 13; disorders of, 669; physiology of, 669.

SLEEP, DISORDERS OF, 669; insomnia, 675; night-terrors, 672; pavor nocturnus, 671; somnambulism, 677.

SMALL-POX. *See Variola.*

SMITH, EUSTACE, on venous hum in functional heart disease, 508; on ve-

nous hum in tuberculosis of lymph-nodes, 384.
Snake venom, a cause of purpura, 532.
Snow, on intestinal intussusception, 220.
"Snuffles," 363.
Sodium benzoate, therapeutic use of, in acetonuria, 567; in enuresis, 602; in influenza, 313; in pseudomasturbation, 609; in valvular heart disease, 504.
Sodium bicarbonate, therapeutic use of, 567; in artificial feeding, 139; in cystopyelitis, 586; in eczema, 722; in endocarditis, 494; in enuresis, 602; in pica, 705; in recurrent vomiting, 256; in rheumatism, 405, 406, 407; in urticaria, 727; in valvular heart disease due to rheumatism, 504.
Sodium chlorid, therapeutic use of, in artificial feeding, 139; in hemoglobinuria, 565.
Sodium citrate, use of, in artificial feeding, 139.
Sodium phosphate, therapeutic use of, in eczema, 722; in enuresis associated with constipation, 603; in purpura, 536.
Sodium salicylate, therapeutic use of, in chorea, 701; in facial paralysis, 666; in pleurisy, 478; in tonsillitis, 422; in urticaria, 728; in valvular heart disease due to rheumatism, 505.
Sodium sulphate, therapeutic use of, in enuresis associated with constipation, 603; in purpura, 536.
Somnambulism, 677; treatment of, 678.
Soper, on use of abdominal belt in whooping-cough, 293.
Southworth, on food percentages, 139.
"Soxhlet's Nährzucker," 125.
Spasm of anus, 236.
Spasmophilic diathesis, 687.
Spanish-American Commission, on incubation period in typhoid fever, 269.
Special senses, development of, 22.
Speech, development of function of, 22.
Spiegelburg, on food consumption in infants, 130.
Spina bifida, 658; definition of, 658; diagnosis of, 660; meningocele form of, 658; meningomyelocele form of, 659; prognosis of, 660; syringomyelocele form of, 660; treatment of, 660.
Spinal column, examination of, in sick child, 31.
Spinal cord, diseases of, 641.
Spine and bony framework, development of, 19.
Spine, curvature of, in school child, 19. gymnastic exercises in correction of, 53.
Spleen, diseases of, 551, 552.
Spleen, enlargement of, 551; in anemias, 522; causes of, 552; clinical significance of, 552; diagnosis of, 551; disorders of malnutrition associated with, 552; in Hodgkin's disease, 541; in acute infectious diseases, 552; in leukemia, 531; in malaria, 282; in mumps, 359; physical signs of, 551; in acute anterior poliomyelitis, 646; in pseudoleukemia, 525; in status lymphaticus, 545, 547; in syphilis, 362, 366, 367; in tuberculosis of mesenteric lymph nodes, 383; in typhoid fever, 271.
Splenocytes. *See Mononuclear leukocytes.*
Splenomegaly, 552.
Spolverini, on development of amylase, 105; on digestive ferments present in milk, 107.
Sporadic cretinism. *See Cretinism.*
Sprue. *See Stomatitis mycosa.*
Squills, objections to use of, in bronchopneumonia, 467; in acute catarrhal bronchitis, 441; in lobar pneumonia, 453.
Staphylococcus vaccine, therapeutic use of, 57.
Starr, table by, showing nature and position of brain tumors, 616.
St. Anthony's dance. *See Chorea.*
Starten's lotion, 723.
Status lymphaticus, 545; anatomy of, 545; anesthesia dangerous in, 547; blood changes in, 547; definition of, 545; diet in, 550; drugs used in treatment of, 550; dyspepsia in, 546; exercise in, 550; fresh air in, 550; mortality in, 548; pathology of, 545; prognosis of, 548; Röntgen rays in diagnosis of, 547; Röntgen rays in treatment of, 548; sudden death in, 546; surgical treatment of, 550; symptomatology of, 546; thymic asthma in, 546; thymic enlargement in, 547; treatment of, 548.
Steel, operation by, for obstruction of esophagus, 158.
Sterilized milk, 119.
Sterility, due to gonococcus vulvovaginitis in infancy, 593.
Still, on orthostatic albuminuria, 569, 570.

STILL'S DISEASE. *See Chronic rheumatoid arthritis.*
STOCK VACCINES, therapeutic use of, 57.
STOOLS, character of, in enteric infection, 191; in fat indigestion, 185; in acute intestinal indigestion, 184; in protein indigestion, 186; in sugar indigestion, 186; therapeutic value of examination of, in illness, 34.
STOMACH, DISEASES OF, 158.
STOMACH WASHING. *See Lavage.*
STOMATITIS APHTHOSA, 147; complicating measles, 338; treatment of, 148.
STOMATITIS CATARRHALIS, 146; treatment of, 147.
STOMATITIS GANGRENOSA, 152; treatment of, 153.
STOMATITIS MYCOSA, 148; treatment of, 149.
STOMATITIS ULCEROSA, 150; treatment of, 151.
STRAMONIUM, inhalation of, in treatment of asthma, 712.
STRAPPING CHEST, for relief of pain in pleurisy, 478.
"STRAWBERRY TONGUE," 321.
STREPTOCOCCUS ENTERITIDES, as cause of enteric infection, 178.
STREPTOCOCCUS VACCINE, therapeutic use of, 58.
STROPHANTHUS, therapeutic use of, in bronchopneumonia, 466; in acute cardiac dilatation, 498; in diphtheria, 307; in acute endocarditis, 494; in intestinal hemorrhage of typhoid fever, 278; in lobar pneumonia, 452; in measles, 340; in meningococcic meningitis, 638; in pleurisy, with effusion, 478; in scarlet fever, 330; in valvular heart disease, 505; in variola, 349; in whooping cough, 294.
STRYCHNIN, therapeutic use of, in asphyxia neonatorum, 73; in bronchopneumonia, 406; in constipation, 208; in acute cardiac dilatation, 498; in diphtheria, 307; in enuresis, 603; in multiple neuritis, 664; in acute pericarditis, 513; in acute anterior poliomyelitis, 651; in scarlet fever, 330; in valvular disease of the heart, 505.
ST. VITUS' DANCE. *See Chorea.*
SUGAR, acid fermentation, due to, in food, 125; indigestion due to excess of, 135, 186; proportion of, in buttermilk, 122, 123; proportion of, in Finkelstein's milk, 123; proportion of, in milk, 103; proportion of, in skimmed milk, 124.
SUGGESTION, therapeutic use of, in epilepsy, 695; in hysteria, 708.
SULPHATE OF SODA. *See Sodium sulphate.*
SULPHUR OINTMENT, therapeutic use of, in congenital ichthyosis, 731; in impetigo contagiosa, 732; in scabies, 736; in tinea tonsurans, 733; in tuberculosis, 729.
"SUMMER COMPLAINT," 180.
SUPPOSITORIES, RECTAL. *See Rectal suppositories.*
SVEHLA, on theory as to cause of status lymphaticus, 545, 546.
SYDENHAM'S CHOREA. *See Chorea.*
SYPHILIS, 360; acquired form of, 360, 361; blood changes in, 366; bones affected by, 360, 361; brain affected by, 366; brain tumor due to, 617; comparative value of mercurial preparations in treatment of, 371; congenital form of, 360, 361; coryza in, 363; deafness from, 367; definition of, 360; diagnosis of, 367; diet in, 369; drugs used in treatment of, 370; etiology of, 361; general malnutrition in, 365, 367; gummatous ulcerations in, 367; hydrocephalus due to, 619, 620; interstitial keratitis due to, 367; iodin in treatment of, 371; kidneys affected by, 366; laryngismus stridulus due to, 685; late hereditary form of, 366; local treatment of, 373; medical treatment of, 370; mercury internally in, 370; mercury by inunction, 370; method of obtaining blood for examination in, 368; microorganism in, 360; myelitis due to, 655; in new born, 83, 364; nose and throat affected by, 367; in nursing infants, 112; pathology of, 362; a predisposing cause of malaria, 594; prognosis of, 368; prophylaxis of, 369; salvarsan in treatment of, 372; skin lesions in, 364, 367; spleen affected by, 366, 367; symptomatology of, 362; teeth affected by, 46, 365; transmission of, 361, 362; treatment of, 369; vaccination not a means of transmission, 353; Wassermann reaction in diagnosis of, 368.
SYRINGOMYELOCELE, 660.
SYRUP OF HYDRIODIC ACID, therapeutic use of, in asthma, 713; in chronic bronchitis, 442; in convalescence from bronchopneumonia, 469; in convalescence from acute laryngitis, 433; in convalescence from lobar pneumonia, 453.

Syrup of iodid of iron, therapeutic use of, in tuberculosis, 399.
Syrup of ipecac, therapeutic use of, in asthma, 712; in acute catarrhal bronchitis, 441; in acute laryngitis, 432; contraindicated, in bronchopneumonia, 467; in lobar pneumonia, 453.
Systemic intoxication, a cause of obscure fever in infancy, 261, 262.
Syrups, objections to use of, 40, 340.

T

Tachycardia, paroxysmal, 506.
Tachypnea, in acute cardiac dilatation, 497.
Talbot, on microscopical examination of stools, 35.
Tapeworm. *See Tenia.*
Tar, therapeutic use of, in eczema, 723.
Tartar-emetic, objections to use of, in bronchopneumonia, 467; in acute catarrhal bronchitis, 467.
Taste, sense of, in new-born child, 22.
Teachers, necessity for knowledge of child's nervous system by, 6.
Teeth, care of, 146; Hutchinson's, 146, 366; imperfection of, in sporadic cretinism, 554; permanent, 146; syphilitic, 365; temporary, 144.
Temperature, of bath in early infancy, 3; of bath in scarlet fever, 329; of cold pack, 405; of incubator, 66; of Priessnitz applicator, 451; of sponge bath in illness, 45; of tub bath in illness, 45, 267; of warm bath in asphyxia neonatorum, 73; of warm bath in eclampsia, 682.
Temperature of room, in acute articular rheumatism, 441; in bronchopneumonia, 468; in acute catarrhal bronchitis, 441; in acute laryngitis, 443; in lobar pneumonia, 450; in measles, 339; in acute nephritis, 581; in scarlet fever, 329.
Temperature, range of, in adenitis, 543; in appendicitis, 225; in acute articular rheumatism, 403; in brain abscess, 618; in bronchopneumonia, 457, 458; in cholera infantum, 192; in chorea, 700; in cystopyelitis, 585; in diphtheria, 297; in empyema, 472; in acute endocarditis, 490; in enteric infection, 190; in erythema infectiosum, 343; in erythema multiforme, 730; in acute gastric indigestion, 159; in acute gastritis, 160; in acute gastroduodenitis, 165; in gonococcus vulvovaginitis, 592; in hemophilia, 538; in Hodgkin's disease, 541; in Holt's inanition fever, 88; in influenza, 309, 311; in acute intestinal indigestion, 184; in chronic intestinal indigestion, 199; in intestinal intussusception, 221; in acute laryngitis, 430; in lobar pneumonia, 445; in lymph-node tuberculosis, 383; in malaria, 281; in mastoiditis, 717; in measles, uncomplicated, 336; in measles, complicated with bronchopneumonia, 337, 338; in meningococcic meningitis, 633; in mumps, 359; in myelitis, 653; in acute nephritis, 577; in otitis media, 714; in pericarditis, 510; in peritonitis, 230; in pleurisy, 472; in acute anterior poliomyelitis, 644; in pseudoleukemia, 525; in purpura hemorrhagica, 533; in recurrent vomiting, 253; in retropharyngeal abscess, 428; chronic rheumatoid arthritis, 408; in acute rhinitis, 412; in rubella, 341; in scarlet fever, 318; in sepsis of the newborn, 78; in septic endocarditis, 494; in stomatitis gangrenosa, 153; in tetanus neonatorum, 90; in tetany, 689; in acute follicular tonsillitis, 419; in tuberculous bronchopneumonia, 462; in tuberculous meningitis, 627; in typhoid fever, 269, 270, 271, 272; in vaccinia, 352; in variola, 346; in varicella, 355.
Temperature, subnormal, in congenital atelectasis, 74; in premature infants, 62; in sporadic cretinism, 554, 555.
Tenia, 211; diagnosis of, 213; differential diagnosis in, 211; elliptica, 213; saginata, 211; solium, 211; symptomatology of, 213; treatment of, 214; varieties of, 211.
Testicle, undescended, 297.
Tetanus, antitoxin of, 59; transmission of, by vaccination, 353.
Tetanus neonatorum, 89; treatment of, 90.
Tetany, 686; definition of, 686; differential diagnosis in, 689; etiology of, 686; pathology of, 687; symptomatology of, 687; treatment of, 689.
Therapeutics of infancy and childhood, 38.
Thermogenic centers, activity of, in young child, 26, 260; loss of restraint over, in heat-stroke, 262; situation of, 25.
Thermoinhibitory centers, 26; in premature infant, 62.
Thigh friction. *See Pseudomasturbation.*

THOMPSON'S SOLUTION OF PHOSPHORUS, 245.
THOMPSON, on congenital laryngismus stridulus, 437.
THREAD WORM. *See Oxyuris vermicularis.*
THROAT AND NOSE, inspection of, in illness, 29; daily disinfection of, protection against endocarditis, 493.
THRUSH. *See Stomatitis aphthosa.*
THUMB-SUCKING, 703.
THYMUS GLAND, anatomy of, 545; asthma associated with enlargement of, 546; enlargement of, in status lymphaticus, 545, 657, 648; excessive activity of, 548; function of, 545; hyperplasia of, 545; surgical treatment for enlargement of, 550; X-ray treatment for enlargement of, 548.
THYROID GLAND, absence of, 557; excessive activity of, 557; excessive activity of, at puberty, 17; insufficient activity of, 555; insufficient activity of, a cause of enuresis, 599; therapeutic use of, 556.
THYROID INTOXICATION, 557; treatment of, 558.
THYROID THERAPY, 554.
TIC. *See Habit spasm.*
TINEA TONSURANS, 733; treatment of, 734.
TONGUE, appearance of, in scarlet fever, 321; appearance of, in typhoid fever, 272, 276; ulceration of, in whooping cough, 289; wandering rash of; *See Geographical tongue.*
TONGUE-TIE, 156.
TONSILS, DISEASED, anatomy involved in, 417; association of, with chorea, 701; association of, with chronic heart disease, 503; association of, with leukemia, 531; association of, with pavor nocturnus, 672, 675; association of, with status lymphaticus, 545, 547; danger from, to health, 415; microorganisms present in, 417; operation for removal of, 421; as portals of infection, 418, 493; removal of, a protection against endocarditis, 493; removal of, a protection against tuberculosis, 395; removal of, in treatment of acute rhinitis, 414; removal of, in treatment of chronic rhinitis, 415.
TONSILLITIS, ACUTE FOLLICULAR, 419; differential diagnosis of, 420; symptomatology of, 419; treatment of, 222.
TONSILLITIS, 418; accompanying adenoid disease, 425; accompanying acute articular rheumatism, 404; accompanying influenza, 310; accompanying acute anterior poliomyelitis, 644; diagnosis between, and diphtheria, 420; differential diagnosis of, 420; acute follicular, 418; preceding laryngitis, 430; prognosis of, 420; recurrent vomiting associated with, 503; acute rhinitis associated with, 413; treatment of, 422; ulcero-membranous, 419.
TONSILLAR HYPERTROPHY, CHRONIC, 420; treatment of, 424.
TORTICOLLIS, INTERMITTENT SPASMODIC, 283.
TOXEMIA, INTESTINAL, in causation of fever, 261, 265; constipation associated with, in typhoid fever, 272; effects of, in diphtheria, 297; effects of, in influenza, 310; effects of, in typhoid fever, 269.
TOXIC NEPHRITIS. *See Nephritis.*
TRACHEOTOMY, in edema of larynx, 435; for relief of foreign bodies in larynx, trachea, or bronchi, 437; in laryngeal diphtheria, 305; in acute rhinitis in infants, 415; in variola, 349.
TRANSFUSION OF BLOOD, in hemorrhage of new-born, 84.
TRICHURIS TRICHIURA, 217.
TRICUSPID REGURGITATION, 502.
TRUDEAU, on treatment of localized forms of tuberculosis, 58.
TROUSSEAU, on causes of tetany, 687.
TROUSSEAU'S SYMPTOM, 688, 689.
TUBERCULIDES OF SKIN, 384.
TUBERCULIN REACTIONS, conjunctival, 385; in diagnosis of concealed lymph-node tuberculosis, 385; in differential diagnosis between tuberculosis and Hodgkin's disease, 542; skin, 36; value of, at different ages, 400.
TUBERCLE BACILLUS, bacteriology of, 373; in pleurisy, 470; in cystopyelitis, 584.
TUBERCULOSIS, 373; of bones and joints, 381, 401; climate in treatment of, 396; contagiousness of, 373; contraindication to nursing infant, 112; cough in, 383, 389; diagnosis between, and early hereditary syphilis, 367; diagnosis between, and Hodgkin's disease, 541; diagnosis between, and malaria, 284; diagnosis between acute miliary, and typhoid fever, 275; diet in, 397; drugs used in treatment of, 398; etiology of, 373; exercise in treatment of, 398; exposure to, 374; fresh air in treatment of, 396; guaiacol inunctions in treatment of, 398; heredity in transmission

of, 375; hydrocephalus due to, 619; acute infectious diseases a predisposing cause of, 375; influenza a cause of, 315; influenza complicated by, 312; intestinal form of, 227, 380; of kidney, 595; of lungs in older children, 388, 401; lymph-node form of, 377, 382; measles complicated by, 337; medical treatment of, 398; general miliary, 381; general miliary in infants, 387; microorganism in, 373; obscure fever due to, 364; pathology of, 377; peritonitis due to, 381, 401; physical signs in, 384; pleural form of, 379; portals of infection in, 374; poverty a predisposing cause of, 375; prophylaxis of, 393; pulmonary form of, 379; removal of cervical glands in, 400; rest in treatment of, 398; school infection in transmission of, 376; as sequel to lobar pneumonia, 448; of serous membranes, 373; of skin, 384; sources of infection in, 373; sputum a source of danger in, 373; symptomatology of, 382; tests for, 36; treatment of, 395; vaccination not a means of transmission in, 355; whooping-cough complicated by, 288, 289, 290.

TUBERCULOSIS OF LYMPH-NODES, cervical form of, 400; cervical adenitis in, 386; diagnosis of, 385; diagnosis between, and Hodgkin's disease, 542; dyspnea in, 382; dwarfishness in, 383; enlargement of external lymph-nodes in, 384; gastrointestinal disturbance in, 383; groups of glands involved in, 378; malnutrition in, 383; menstrual irregularities in, 383; neurotic disease suggestive of, 382; pain in side in, 382; pathology of, 377; physical signs of, 384; pressure signs of, 385; progressive failure of health in, 382; radiographic examination in, 385; removal of glands in, 400; respiratory symptoms in, 383; simple anemia suggestive of, 382; skin manifestations of, 384; temperature in, 383; tuberculin reactions in, 385.

TUBERCULOUS BRONCHOPNEUMONIA, 387; treatment of, 401.

TUBERCULOUS MENINGITIS. *See Meningitis, tuberculous.*

TURPENTINE, OIL OF, inhalations of, in treatment of acute rhinitis, 414.

TYPHOID FEVER, 267; abdominal symptoms in, 271; antityphoid inoculation in, 276; bacillus of, 267; baths in treatment of, 277; blood changes in, 273; "carriers" of, 267, 268; complications of, 274; definition of, 267; differential diagnosis in, 275; diet in, 276; drugs used in treatment of, 277, 278; epistaxis in, 273; etiology of, 267; exanthem in, 271; "carriers" of, 267, 268; in fetus, 269; incubation period in, 269; intestinal hemorrhage in, 272, 278; meningitis in, 640; mortality in, 275; method of infection in, 268; nervous system in, 272, 278; in new-born, 269; obscure fever due to, 265; pathology of, 268; perforation of intestine in, 272; prophylaxis of, 275; prognosis of, 275; relapses in, 274; removal of patient in, 279; respiratory symptoms in, 273; sources of infection in, 268; splenic enlargement in, 271; symptomatology of, 269; temperature changes in, 270; tongue in, 272; treatment of, 276; urine findings in, 273; Widal reaction in diagnosis of, 273.

TYPHOID VACCINE, therapeutic use of, 59.

TYROTOXICON, symptom group produced by, 176.

U

ULCER OF STOMACH, 164.

UMBILICAL CORD, CARE OF, 3.

UMBILICUS, DISEASES OF, 84; gangrene, 85; hemorrhage, 85; hernia, 86; infection of stump, 84; vegetations, 84.

UNGUENTUM CREDÉ, therapeutic use of, 43; in adenitis, 544; in dermatitis exfoliativa of new-born, 80; in erysipelas of new-born, 82; in measles, 340; in acute infective myelitis, 657; in septicemia of diphtheria, 307; in septicemia of scarlet fever, 331; in tuberculous peritonitis, 401; in ulcerative endocarditis, 494.

UPPER ARM PARALYSIS, 99.

UREA, amount of, in infancy, 561; retention of, in acute nephritis, 577.

UREMIA, presence of, in cystic degeneration of kidney, 589; in acute nephritis, 576; in tumor of the kidney, 588; eclampsia caused by, 680.

URETHRITIS, IN MALE CHILD, 595.

URIC ACID, "infarcts," 561.

URIC ACID, in urine, in infancy, 561; in acute nephritis, 577; in whooping-cough, 290.

URINE, acidity of, a cause of pseudo-masturbation, 607, 609; effect of uric acid upon, 561; examination of, in illness, 34; fluctuations in amount of, 560;

frequency in passage of, 561; incontinence of, 561; method of collecting for examination in infant, 559; retention of, in myelitis, 656; suppression of, in absence of kidney, 577; suppression of, in acute nephritis, 577; suppression of, in new-born, 561; suppression of, in young child, 560.

UROGENITAL ORGANS, embryonic development of, 604; nerve supply of, 605; physiological evolution of, 605.

UROTROPIN, therapeutic use of, in cystopyelitis, 587; in enuresis, 603; in mumps, 360; in acute anterior poliomyelitis, 651.

URTICARIA, 726; accompanying asthma, 713; characteristic of, 726; definition of, 726; diet in, 728; etiology of, 726; accompanying Henoch's purpura, 534; mucous membranes in, 727; prognosis in, 727; accompanying purpura rheumatica, 535; symptomatology of, 726; treatment of, 727.

UVULA, ELONGATED, 155.

V

VACCINIA, 349; clinical manifestations of, 351; complications of, 353; definition of, 349; incubation period of, 349; temperature in, 351; secondary rash in, 352.

VACCINATION, discovery of, 349; immunity, conferred by, 353; technique of, 350; treatment of wound, 354; virus used in, 350.

VACCINE THERAPY, 55; antiserums in, 59; autogenous vaccines in, 57; bacterial vaccines in, 56; clinical reaction in, 56; coli vaccines in, 59; in cystopyelitis, 587; discovery of, 55; in chronic empyema, 481; in furunculosis, 729; gonococcus vaccine in, 58; in gonococcus vulvovaginitis, 594; Koch's tuberculin in, 58; in lobar pneumonia, 452; opsonic index in, 56; pneumococcus vaccine in, 58; rules for use of, 56; in scarlet fever, 332; staphylococcus vaccine in, 57; streptococcus vaccine in, 58; therapeutic indications for, 57; typhoid vaccines in, 59; in ulcerative endocarditis, 494; in whooping-cough, 294; without opsonic index, 56.

VALENTINE'S MEAT JUICE, 277.

VALERIAN, therapeutic use of, in asthma, 708; in functional heart disorders, 508.

VARICELLA, 354; blood changes in, 356; complications in, 356; definition of, 356; diagnosis of, 354; diet in, 357; drugs used in treatment of, 356; enanthem stage in, 355; etiology of, 354; exanthem stage in, 356; immunity conferred by, 357; incubation period in, 355; microorganisms present in, 354; prophylaxis in, 357; quarantine in, 357; symptomatology of, 355; temperature changes in, 355; treatment of, 357.

VARIOLA, 345; blood changes in, 347; clinical forms of, 347; confluent form of, 347; contagious period of, 345; definition of, 345; diagnosis of, 348; diet in, 348; drugs used in treatment of, 348, 349; enanthem stage in, 346; etiology of, 345; exanthem stage of, 346; hemorrhagic form of, 347; immunity conferred by, 345; incubation period of, 345; invasion stage of, 345; microorganisms present in, 345; mortality from, 350; prophylaxis of, 348; purpuric form of, 348; quarantine in, 348; red light treatment of, 349; symptomatology in, 345; temperature changes in, 346; transmission of, 345; treatment of, 348; urine findings in, 347; vaccination as protection against, 349.

VASOMOTOR PARALYSIS. *See Splanchnic paralysis.*

VAUGHN, on contaminated food, 176.

VEGETATIONS OF THE UMBILICUS, 84.

VENESECTION, therapeutic use of, in acute cardiac dilatation, 498; in eclampsia, 683; in acute nephritis, 580.

VERONAL, therapeutic use of, in chorea, 701; in meningococcic meningitis, 638; in multiple neuritis, 664; in acute nephritis, 581.

VILLEMIN, operation by, for obstruction of esophagus, 158.

VINCENT'S ANGINA. *See Ulceromembranous tonsillitis.*

VINCENT'S BACILLUS, 419.

VINCENT'S SPIRILLUM. *See Vincent's bacillus.*

VIPOND, isolation by, of bacillus producing scarlet fever, 317.

VLEMINCKX'S SOLUTION, formula for, 729.

VOMITING, as symptom, of brain abscess, 618; of brain tumor, 618; of enteric infection, 191; of intestinal intussusception, 220; in lobar pneumonia, 443; in malaria, 284; in meningococcic meningitis, 632; in purulent meningitis, 639; in recurrent vomiting, 283; in

scarlet fever, 318; in tuberculous meningitis, 627.
VOMITING, RECURRENT. *See Recurrent vomiting.*
VON JAKSCH, discovery of pseudoleukemia by, 524.
VON JAKSCH'S DISEASE. *See Pseudoleukemia.*
VON NOORDEN, on factors in formation of oxybutyric acid, 565.
VON PIRQUET, on predisposing causes of tetany, 687; on scarification tuberculin test in diagnosis of lymph-node tuberculosis, 385; on skin reaction in tuberculous meningitis, 628, 629.
VON PIRQUET'S TEST, for tuberculosis, 35.
VOVAIRD, investigation by, on primary splenomegaly, 552.
VULVOVAGINITIS, GONORRHEAL, 591; complications of, 592; definition of, 591; epidemics of, 591; etiology of, 591; frequency of, 595; microorganisms in, 592; prognosis of, 593; prophylaxis of, 593; sources of infection in, 591; symptomatology of, 592; treatment of, 593; vaccine therapy in, 594.
VULVOVAGINITIS, SIMPLE, 595; association of, with pseudo-masturbation, 608.
VULVA, DIPHTHERIA OF, 299.

W

WALKER-GORDON LABORATORIES, 141.
WALKER-GORDON MILK, 118.
WANDERING RASH OF THE TONGUE. *See Geographical tongue.*
WARTHIN, on edema of thymus gland, 545; on weight of thymus gland in health and disease, 545.
WASSERMANN-NEISSER REACTION, 362, 368.
WATER, DRINKING, in causation of typhoid fever, 268, 275; importance of, in infant diet, 105; importance of, to new-born, 113.
WATER, DRINKING, therapeutic use of, 47; in influenza, 313; in lobar pneumonia, 451; in multiple neuritis, 663; in chronic nephritis, 583; in orthostatic albuminuria, 571; in purpura, 536; in typhoid fever, 277.
WEANING, 116.
WEICHSELBAUM, discovery by, of special organism of meningococcic meningitis, 631.
WEIGHT, comparative ratio of, in boys and girls, 16, 17; gain in, during first year of life, 15; gain in, after first year of life, 15; importance of ascertaining, at first examination in illness, 29; importance of, to health, in infancy and childhood, 13; premature infants deficient in, 65; relation of, to physical condition, 7; relation of, to skin surface of body, 26; significance of failure to gain in, 14.
WELSH, E. J., method of injecting blood serum introduced by, 84.
WET NURSE, advantages of, 117; in gastrointestinal disorders, 74; in enteric infection, 194; for premature infant, 68; in pseudo-leukemia, 526.
WET NURSE, danger to, of syphilitic infant, 369; of tuberculous infant, 394.
WHEY, as artificial food in chronic intestinal indigestion, 201; proteins contained in, 101.
WHISKEY, therapeutic use of, in bronchopneumonia, 466; in acute cardiac dilatation, 498; in diphtheria, 307; in enteric infection, 193; in lobar pneumonia, 450, 453; in new-born, 73, 81; in acute pericarditis, 513; in pleurisy, 478; in acute anterior poliomyelitis, 651; in scarlet fever, 329; in typhoid fever, 277, 278; in variola, 349.
WHOOPING-COUGH, 287; bacillus of, 287; blood changes in, 289; catarrhal stage in, 288; complications of, 290; contagious period in, 288; course of, 290; acute catarrhal dilatation caused by, 496, 497; danger of, to infants, 6; danger of, to tuberculous children, 395; definition of, 287; diagnosis of, 290; diet in, 292; drugs used in treatment of, 293; etiology, 287; incubation period of, 288; measles complicated by, 338; medical treatment of, 293; prognosis of, 291; prophylaxis in, 291; psychic treatment of, 292; spasmodic stage of, 288; symptomatology of, 288; temperature changes in, 288; treatment of, 291; urine findings in, 290.
WICKMAN, on mortality in acute anterior poliomyelitis, 650; on types of acute anterior poliomyelitis, 644.
WIDAL REACTION. *See Gruber-Widal reaction.*
WILLIAMS, on thyroid insufficiency as a cause of enuresis, 599; on thyroid therapy in enuresis, 603.
WINCKEL'S DISEASE. *See Epidemic hemoglobinuria.*

WINTERGREEN, OIL OF, therapeutic use of, by inunction, 42, 43; in acute follicular tonsillitis, 422.
WOLLSTEIN, observations by, on *bacillus pertussis*, 287.
WRIGHT, E. A., on antityphoid inoculation, 276; on "opsonins," 55, 56.
"WRIST-DROP," 662.

X

X-RAY. *See Röntgen rays.*

Z

ZINC, multiple neuritis due to poisoning by, 661.
ZINC OXID, therapeutic use of, internally in tuberculous diarrhea, 400.
ZINC OXID, OINTMENT OF, therapeutic use of, in eczema, 723; in erythema multiforme, 730; in syphilis, 373.
ZINC STEARATE, therapeutic use of, in eczema, 723; in pemphigus neonatorum, 733.

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